# RELATIONSHIP BETWEEN DIET AND DENTAL CARIES[1]

E. V. McCOLLUM, PH.D., Sc.D.

*School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Maryland*

I appreciate the opportunity of being here this evening to give such thoughts as I have on the relation of diet to dental caries. What I shall say will be mainly an interpretation of the results of animal experimentation as they relate to this subject. Not many years ago the slogan was frequently heard that a clean tooth never decays. This was given wide publicity and the promise was made that assiduous application of a tooth brush with a recommended dentifrice would insure against tooth decay. That idea has been discredited. Advice on prophylaxis through diet, as well as advice concerning how to keep the mouth clean, has frequently failed to accomplish its purpose.

There are people in various parts of the world who enjoy relative freedom from tooth decay. The northwest Greenlanders exist essentially upon a carnivorous diet, consisting principally of eggs and the flesh of birds, the beluga whale and the norwal. Only for a short time during the summer do they get any fish. They get almost no land animals. They are said by Dr. Hrdlicka to have the best teeth of any fairly large group of people in the world today. They are nearly free from dental disease. On the other hand, there are people adhering strictly to a vegetarian diet, or a vegetarian diet supplemented with milk, who likewise have relatively little dental disease. I have come in contact in the last fourteen years with a considerable number of foreign students. The Hindu students, mainly medical men past thirty years of age, have in general distinctly better teeth than have Americans.

A recent report on the condition of the teeth of the inhabitants of the Islands of Tristan da Cunha by Dr. Marshall affords an interesting illustration of the effect of the character of the diet on the health of the

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teeth. These islands are 1500 miles west of the Cape of Good Hope, and about 4000 miles east of Cape Horn. About 140 people live on these small islands. Before the time of steam vessels for hunting whales in the South Seas, sailing vessels frequently stopped there for supplies and provided the inhabitants with some food from the outside world. With the advent about two generations ago of steam trawlers the whalers became independent of the islands and the natives have rarely seen any one from the outside world for fifty years. The islands are so badly infested with rats that the natives have been unsuccessful in the cultivation of cereal grains. They grow onions, cabbage, carrots, turnips and beets. The principal food is eggs and the young of guillemots. They collect thousands of both birds and eggs during the summer and preserve them for food throughout the year. They use almost no beef, although they have cattle. They have some milk and a small amount of fish, although fish are abundant. They have had no sugar, jams or sweets of any kind for two generations. Dr. Marshall examined the teeth of 54 of the 141 people on the island in 1928, and found that 13 in the age group of 3 to 14 years showed collectively only 2 carious teeth; 8 people, aged 15 to 20, showed no caries; 21 people of 21 to 44 years showed collectively 11 carious teeth; and 12 people, 45 to 90 years of age, had collectively 21 carious teeth. These people, who have eaten no sugar, have likewise eaten relatively little cooked starch.

Dr. Cilento, with whom I have been in correspondence for a number of years, has been a close observer of the people of the islands off the coast of New Guinea. These people were formerly well developed and healthy, but in the last few decades all valuable farm land has mainly fallen into the hands of foreigners, and now the natives are in a miserable state and subsist largely upon starchy roots. Swamp taro, yams, three kinds of breadfruit and bananas constitute almost their only food, although they eat some fish and a few birds and lizards. Their nutrition is poor. Their mouths are very unclean, their teeth highly colored, and decay and apical abscesses are very common. They lose a great many of their teeth by the time they reach middle age.

We must be convinced from these examples that there are vegetarians, carnivorous people, and people on various types of mixed diets,

who according to reports probably have better teeth than are found among Americans. How can we account for the observed defects? Dr. Wallace of Edinburgh has insisted that dental decay and caries are independent of all dietary factors other than the consumption of sugar, and his view is that there is no significance in the developmental factor, that is, the structure of the teeth in relation to dental disease. He points out that the people on some of the islands off the coast of Scotland who eat freely of cereals, particularly oats, and who eat a good deal of fish, but who have no sweets, are nearly free from dental caries, although they eat much cooked starch, whereas the same stock of people in industrial districts in Scotland have a very high incidence of dental decay.

Those who have devoted attention to research on diet now visualize an adequate diet in terms of perhaps thirty-five or thirty-six chemical substances. Further research will probably show that several additional nutrient principles are necessary. We do not know for certain how many of the digestion products of proteins are indispensable in the food; but, for the purpose of illustrating what constitutes an adequate diet, I will assume that seventeen of these products can not be synthesized by the body. In addition to these protein-digestion products we must have a source of glucose in the form of starch or sugars which are convertible into glucose during digestion. We are certain that the diet must provide at least ten inorganic elements, namely, sodium, potassium, calcium, magnesium, chlorine, iodine, phosphorus, sulphur, iron and copper. Collectively this makes twenty-eight. We know further that there are at least six substances which we collectively designate as vitamins and differentiate as vitamins A, B, C, D, E and G. These are unique in that the amount of each which we require for normal nutrition is astonishingly small. Yet in the absence of any one of them a specific kind of malnutrition develops. I shall not attempt to describe the different kinds of dietary deficiency diseases, but will speak only of those deficiencies which may play an important rôle in maintaining the health of the teeth and their attaching tissues.

*Vitamin A* is one of the three fat-soluble vitamins. Whenever this is lacking in the diet or is provided in insufficient amount the epithelial tissues everywhere suffer injury. Even the highly specialized

epithelium lining the ducts of the glands, and those cells that produce such characteristic secretion as tears, saliva and other digestion fluids, tend to keratinize and become similar to the outer layer of the skin. This keratinized epithelium tends to desquamate and plaques of it may tend to plug the ducts. This is seen in the lacrymal, harderian and meibomian glands, in the bile duct and in the kidneys and ureters. Such epithelial débris has been suggested as a factor in the development of gall stones and kidney stones. The mucosa of the intestine is also impaired both as to its capacity to secrete digestive fluids and to absorb digestion products in the normal way. Howe and Wolbach have called attention to the fact that when the diet is deficient in vitamin A the odontoblasts revert to osteoblasts, and that the latter may subsequently lay down bone where dentine would normally have been formed. The etiology of pulp "stones" is suggested by this observation. We have no observations tending to show whether in poorly nourished human subjects vitamin-A deficiency may exercise an adverse influence upon the odontoblasts. In other words we do not know how sensitive are the odontoblasts to an under-supply of vitamin A nor what period of partial deprivation would affect them adversely.

It is well known that deprivation of *vitamin B* results in injury to the peripheral nerves, but no one has investigated the nerves in the pulp to ascertain whether these undergo the same changes as do the peripheral nerves generally. Unusual interest attaches to the relation of *vitamin C* deficiency to the health of the teeth. Earlier studies showed that, as scurvy develops, the endothelial tissues are especially susceptible to injury. It is due to this that the capillary vessels break down, causing the hemorrhage characteristic of this disease. It remained for Höjer to demonstrate that the odontoblasts are peculiarly susceptible to injury through deficiency of vitamin C. He showed that, when guinea pigs are fed for 12–14 days a diet containing about 80 percent of the minimum protective dose of the antiscorbutic vitamin, the cells constituting the odontoblastic membrane become uneven in length and present a somewhat serrated margin. When about half of the minimum protective dose is fed, the odontoblastic membrane separates from the dentine, apparently with a detachment of the fibers that penetrate the dentinal tubules and pass to the base of the enamel. When 30 percent of the minimum protective dose of the vitamin is fed,

the odontoblastic layer breaks up into islands of distorted cells. No one has studied the extent to which the odontoblasts may return to normal after a specific amount of injury has been effected by this procedure. Since capillaries are so sensitive to vitamin-C deficiency, there is likely to be hemorrhage in the pulp cells and injury to the odontoblasts. The subject is in great need of further study. The point to be emphasized is that the odontoblasts are so much more sensitive than the capillaries to a deficiency of vitamin C that, in approximately half the time necessary to produce clinical evidences of scurvy through deficiency of the vitamin, injury to these important structures in the pulp has already developed. We need an explanation for the frequency of the occurrence of pulpless teeth. Since Lind showed many years ago that clinical scurvy develops in man after about forty days of total deprivation of fresh, uncooked vegetable food, we may be justified in suspecting that even twenty days of deprivation of vitamin C in an adult would cause injury to the pulp which could be demonstrated histologically.

There can be no reasonable doubt that there is a close relation between deficiency of *vitamin D* in the diet and the structure of the teeth. The work of Mrs. Mellanby on pulps has demonstrated clearly that diets of a character which tend to produce rickets tend also to interfere with the proper calcification and enamelling of the teeth. Vitamin D, as is well known, has remarkable power to regulate calcium and phosphorus metabolism. A suitable provision of it is effective in the prevention of rickets even when dietary conditions are such as in its absence to disturb bone growth.

There are two remaining *vitamins*: *E and G.* The former is concerned with the maintenance of fertility and the latter is the etiological agent in pellagra. Neither of these has been shown to have any peculiar relation to the health of the teeth.

The list of nutrients thus far discussed sum up to thirty-four. Recently Dr. and Mrs. Burr of the University of Minnesota have published experimental evidence, seemingly quite conclusive, which demonstrates that while the body can synthesize saturated fatty-acids such as palmitic and stearic, or a singly unsaturated fatty-acid such as oleic, it fails in the synthesis of doubly and triply unsaturated fatty-acids such as linoleic and linolenic acids. These are structural com-

ponents of protoplasm; and since the body is unable to synthesize them, it appears that at least one, linoleic, must be provided in the diet. This discovery adds a thirty-fifth nutrient principle in the form of a relatively simple chemical compound that is indispensable in the diet. Doubtless the end is not yet, and we may confidently expect that several new substances—inorganic elements, vitamins, etc.—will be shown to be indispensable components of the diet, because the tissues cannot produce them synthetically from some other substance.

I present this picture of the adequate diet to help you visualize the manner in which investigators think about an adequate diet. It will be apparent also that modern research in this field involves the many problems concerning the optimum amount or proportions among these substances, the effect of deficiency or excess of one or another, and a search for new and hitherto unsuspected nutrient principles. If there is any relation between diet and dental caries, and there is a general belief that there is, it must be explainable, when we have sufficient evidence, on the basis of either a deficiency of one or another of the essential nutrients, or of unfavorable quantitative relations between two or more of them.

One is tempted to speculate as to what may follow injury to the odontoblastic membrane owing to deficiency of vitamin C even to an extent that presents no clinical symptoms of scurvy. Each odontoblast has a fibre which permeates a dental tubule; and when the odontoblasts are caused to separate from the dentine, I assume that the dentinal tubules would probably be opened by atrophy of the fibrils which normally permeates them. This should provide an easy access and promote a much greater flow of lymph from the pulp cavity into the dentine than normally takes place, and should perhaps give an opportunity for infection of the tooth by way of the blood stream. It is well known that in normal individuals a few bacteria gain access to the blood stream. The number which enter the blood may be much greater when there is a focal infection or a persistent unhygienic condition of the colon. Bacteriemia has been produced in rats, by Sollman, by feeding them constipating diets. Even though the pulp of a tooth may be the seat of hemorrhage, if an organism should be introduced into the pulp chamber by way of the vascular system, is it not logical to expect that, if the dentinal tubules were open and the barrier of the

odontoblastic layer broken, the tooth might be infected from within. If deprivation of vitamin C should be more pronounced we should expect, if the capillaries in the pulp are as sensitive as other capillaries to this type of deficiency, that a pool of blood would be formed in the pulp. Dr. Howe of Boston has demonstrated with guinea pigs and monkeys that a deficiency of vitamin C is very inimical to the health of the teeth, and that if this deficiency exists in the developmental period, hypoplastic enamel is the result.

It is well known that enamel dissolves readily in acids, and a sheltered area in which fermentable food is retained is, according to the experience of dentists, the area in which decay is likely to occur. Acid on a tooth surface can do little harm where saliva is frequently applied and replaced by new saliva, since this is a buffer solution and can neutralize much acid. To this extent the whole view of caries seems sound, namely, that it is primarily the result of acid disintegration of the enamel, which makes a breach, eventually penetrating the dentine. I do not believe that the development of dental caries is dependent *solely* upon the action of acid. There is too much evidence that certain types of defective diet injure the tooth from within. Mellanby's observations on defective tooth-structure resulting from vitamin-D deficiency are of outstanding significance in this respect. When viewed in the light of the histological studies on bone which Park, Shipley, Simmonds and myself conducted some years ago, they offer convincing evidence that in the development of rickets the organic matrix of bone is abnormal. The marked effect of vitamin-D deficiency on the behavior of cartilage cells, and on the development of osteoid, and on the appearance of the osteoid tissue, when diets differ in their contents of calcium, phosphorus and vitamin D, is easily seen. It is hardly conceivable that the osteoid of bones modified in the ways we described is all of equally good quality for the performance of the functions of that tissue. Similar conclusions must be warranted in connection with the structure and functional capacity of the organic matrix of the tooth. It is for this reason that I believe the defective tooth demonstrated by Mellanby must be important as a predisposing factor in the development of dental disease.

A contribution of unusual interest has been recently made by Hanke. He states that by giving patients enormous doses of citrus-fruit juices,

and therefore a high content of vitamin C, both dental caries and pyorrhea may be resisted in nearly all individuals. His results appear to be explainable only on the assumption that the organic structures in the teeth and around the teeth are functioning abnormally in people exhibiting what is generally regarded as normal health, but who nevertheless are subject to tooth decay. His experience, if explained on this basis, would require the assumption that the great majority of American people are getting too little of the antiscorbutic principle, and that certain tissues are in a borderline state of malnutrition for this reason. Höjer states that the effects of deficiency of vitamin C become "manifest at a rate that varies for the different cells and tissues; first in the odontoblasts, then in the osteoblasts, muscle cells, lymphoid tissues, fibroblast, and salivary glands, then in the liver, adrenal, chondroblasts, and last in the epithelium of the kidney. The order in which the changes set in and continue in odontoblasts, osteoblasts and chondroblasts seems to argue for the notion that phylogenetically older and functionally less differentiated cells are later and more lightly struck by the deficiency of antiscorbutic." I deduce from all the evidence presented that it is probably true that the majority of American people at times take an inadequate amount of vitamin C. It is probable that total deprivation for a period no longer than three weeks leaves its mark on the living structures in the teeth, and that such a tooth is ready for the development of a carious lesion.

If a relation between vitamin C and the development of caries is demonstrated, it must be harmonized with the apparently established fact that certain age groups, especially those of adolescent years, are more prone to suffer from tooth decay following dietary practices which are less satisfactory than are other age groups, where decay less frequently occurs. It would be quite interesting to study the history of individuals who at one time suffer greatly from caries but who with advancing age become relatively or quite immune. An argument against the view that dental caries is so completely associated with vitamin-C deficiency is that the quality of the diet of the Eskimo and of the people of the Islands of Tristan does not seem to warrant the belief that either of these diets is excessively rich in the antiscorbutic principle. Muscle meat is known to be very poor in this substance, fat contains none, egg white none, and the yolk apparently only a very

moderate amount. We do not know how much of the vegetable food eaten by the natives of Tristan is eaten raw. I have not been able to learn to what extent the natives of the Islands about New Guinea eat raw food. They eat freely of bananas. Since this fruit is rich in the antiscorbutic principles it would appear that banana-eating people should be protected to some extent against dental disease. It is of interest in this connection to mention that we have in our experimental rat colony many times produced animals with marked susceptibility to tooth decay, notwithstanding the rat is able to synthesize vitamin C. Animals reared to middle life or beyond on food containing no trace of this substance have livers which are very rich in the vitamin. I am aware of a recent statement by Eddy that in rats the odontoblasts are adversely affected by a diet deficient in vitamin C.

Some years ago Grieves and I described the teeth of numerous experimental rats whose diets were deficient in different principles. A deficiency of calcium and vitamin A together produced the most pronounced effect upon the teeth. From the information which I have as a result of prolonged observation we cannot dismiss the calcium-phosphorus ratio and the provision of vitamins A, C and D in any consideration of the relation of diet to dental disease. Entirely apart from the content of vitamin C in the diet, the bones of rats may be made fragile or densely calcified, and resorption of bone may be brought about, without any consideration of the vitamin-C content of the diet in this species.

In collaboration with Dr. Henry Klein I have for several years been conducting experimental work supported jointly by the American Dental Association, the dental group of the Johns Hopkins Hospital, and the Bureau of Animal Industry, Doctors Howe and Buckley of the Bureau participating in the work. Up to the present time we have limited our experiments to the feeding of swine on diets so constituted as to produce experimental rickets in pigs after the weaning period. We selected swine because of the long gestation period and the omnivorous habits of this species. The results thus far obtained show that the feeding of a rickets-producing diet to sows during the second half of the gestation period of one hundred and fifteen days, although very injurious to the mothers, does not to any appreciable extent adversely affect the teeth of their progeny. When such diets are fed to sows dur-

ing the ten weeks' suckling period, the milk is not of good quality and the teeth and jaws are adversely affected as to structure, position and tendency to succumb to disease. When the rickets-producing diets are fed directly rather than through the mother, at the age of seventy days and beyond, pigs suffer still more marked effects in tooth structure; hypoplastic enamel, open apices, abnormally large pulp cavities and mal-occlusion are among the defects noted. The control group of animals without antiscorbutic food have teeth which are in extraordinary contrast in their perfection to the defective teeth of experimental animals.

While, therefore, I feel that no final conclusions can be drawn at present concerning the etiology of dental disease in its relation to diet, I cannot see how the extreme views of those who place all the emphasis on the antiscorbutic vitamin can be justified.

## DISCUSSION

*Lafayette B. Mendel, Ph.D., Sc.D. (New Haven, Conn):* I have been greatly privileged tonight, and I believe all of you have, to listen to an unusually clear and cogent exposition of our newer knowledge of nutrition; and to have heard presented in a very unusual and stimulating way not only the acquisitions that have come from the recent study of the problems of dental growth and dental disease, but also (and this is a very important point, I think, in every scientific discussion) some of the problems that need to be considered in the near future. You can scarcely expect me, in fact I know you don't expect me, to add anything of significance to the masterful address that we have heard tonight. But perhaps I can take a few minutes to hitch onto it, so to speak, a few considerations regarding the problems of dental research and the outlook for dental research in the immediate future. I feel that the time has come when there ought to be more critique exercised in the research programs that are about to be applied in this large field; and perhaps it will be worth while if we hastily consider some of the more important criteria.

In planning a scientific experiment, a good investigator always has in mind the reduction of the number of variables to a minimum. If he has a half-dozen factors that may vary under the plan of investigation, his delight is to be able to rearrange his program so that only one variable out of the half-dozen will remain; and then from the result, with the variable either included or left out, he may draw a cogent conclusion. It happens, however

that there are relatively few instances in the study of nutrition in which plans of experimentation can be so elegantly outlined. You will realize, if you recall the significant suggestion of Dr. McCollum, that there are at least thirty-five simple factors to be considered in an adequate diet. To plan investigations dealing with the relation of diet to pathology, or to normal or abnormal growth, therefore, is a very serious problem from the standpoint of those variables alone. In the past a great deal of the inadequacy of the programs of investigation has lain in the fact that there have been too many variables. This is one of the features of research that ought to be modified, or improved at the earliest possible opportunity. To give you one or two illustrations that occur to me on the spur of the moment: we have learned a great deal about how to feed certain laboratory animals, the white rat for example. It was only a short time ago that we began to realize that these animals often secure one of the essentials through the eating of their feces; if they are deprived of certain elements, they conserve this material in a way by the habit of eating their stools. It was only by rearranging the experimental conditions in such a way that the animals were unable to reach the feces (through the use, for example, of false bottom cages) that the dosage problem in connection with some of the vitamins was very materially altered. That is a very little thing, but it represents a variable that hadn't been considered before. I could go on with further illustrations, but that is enough perhaps to stress the point I have in view.

When we come to experiments on groups of humans—and these are becoming very popular in the field of dental research; taking groups of a few hundred children in an orphanage or groups in a school—we really learn very little about them. We know very little except in a very casual way about the diets of such children day in and day out, not to say very much about all the many other variables, the external ones. You can see how extremely difficult it is going to be, or how difficult it has already been, to draw any cogent conclusions from such a heterogenous series of external factors. I include diet in the external factors, such as involve groups of school children or groups of individuals in institutions here and there. I think this very fact has been responsible for the circumstances that, as the result of such types of investigation, one is told in one city to eat green vegetables to avoid a certain dental effect. In Chicago, now, you drink citrus-fruit juices. Go to another city and you use codliver oil. You go somewhere else and another device may be under consideration.

I doubt whether some of you at least realize how great the difficulties are in securing really dependable statistics from groups of the sort mentioned, in contrast to what can happen in the case of animal experimenta-

tion of the sort Professor McCollum detailed to you this evening. When you are dealing with groups of persons, it is imperative, in order to get any conclusions worth anything whatever, to deal with the groups largely statistically. I might mention many illustrations to show the difficulties that arise in all kinds of research where one is dealing with statistical methods, and the tremendous numbers of unknown things that must enter into consideration. What is the solution of such a difficulty? One of the solutions is that we must have better subjects of research, research animals, if you please. The human individual is, for reasons that we have just mentioned, one of the most difficult of all to deal with. If you will recall what the discovery of an experimental animal that is subject to scurvy has meant for our knowledge of that disease, you will have the point clearly before you. Dr. McCollum called your attention a few moments ago to the fact that scurvy has been known for 175 years as a clinical disease. It was not until only a few years ago, relatively speaking, that the discoveries of Scandinavian investigators disclosed that the guinea pig is a particularly suitable animal to evidence the manifestations of scurvy under certain conditions. From this our modern knowledge of scurvy, of antiscorbutic foods and vitamin C, as we call it now, was adequately developed. It was the discovery of a suitable experimental animal that spelled success. We have learned about the distribution and the character of vitamins, and the meaning of those interesting factors in nutrition, through the availability of an experimental animal that Dr. McCollum has used so very successfully in his classic researches, the albino rat. Think what that has meant for us.

So, gentlemen, I feel that one of the great desiderata in scientific dental research is to find an experimental animal other than man. I think, also, after what we have heard regarding the peculiarities of the dentition of the rodents and particularly of the rat, some animal other than the rat, which is so widely used as an experimental subject at the present time, should be used. We have this need of a suitable experimental animal. Shall it be the dog? Shall it be the cat? Shall it be the monkey or the primate or the pig, or some other species that I haven't in mind at the moment? When dental investigators have discovered through the study of the normal dentition the characteristics of dental growth, et cetera, of various species, and have selected a proper subject for experimentation other than man, I feel that there will be a better opportunity than has ever been developed hitherto for the study of some of these problems that have been touched on so interestingly this evening. I think, too, that the time has come, with this tremendous awakening of interest in the problems of scientific dentistry, with the development of a great group of young men in this country who are

trained to approach the problems of their profession in a new way compared to what was true a generation ago, for insistence upon a far greater degree of critique than has been characteristic of a large part of the profession in the years gone by. It has probably been true (and this is not said by way of disparagement), largely because of the relatively slow development of the dental profession in its scientific aspects, that dentists as a group have been more inclined than they ought to have been to accept large numbers of statements at their face value without further consideration. Dogmatism has been one of the despairs and the detriments of the development of dentistry. The time has come to take a Missourian attitude in all of the things that you have to approach in your profession precisely as the medical profession has slowly learned that it must do. We have been in the past too often "educated beyond our knowledge;" a condition that it is very important to avert.

If I were going to advise (it is a dangerous thing to do) regarding the attitude that ought to be developed in the young men who are coming into the field of dental research in the next few years, I should stress perhaps three points and leave them with you as a parting thought. There ought to be developed in those young men an *open mind*. Inertia is one of the banes in the attempt to make progress in the scientific world. Inertia is of two sorts. One can be smug and entirely satisfied with things. People who don't care about change represent a kind of inertia. They don't have an open mind. Others are prone to act but never deviate from a definite direction. That is also an inadvisable sort of inertia. We must develop in our younger people a *respect for facts*. That is what one learns in the world of business, in the development of a machine, in the construction of a building. All of that is based on facts. There is no guesswork there; there is no hypothesis. From the world of business we can learn respect for facts. Business doesn't always have an open mind, though. The third point is that which Pasteur stressed so widely; that is the importance of having a *prepared mind*, a mind that is so receptive to current information or older information, that when the opportunity comes, no matter where it is, the individual will be prepared to profit by it. It doesn't make any difference whether you are living in the city of New York or are in the wilds of a western state, if you have the prepared mind and you are a dentist, you can benefit by all sorts of opportunities. One of the finest auguries for the future of scientific dentistry, and the advancement of the profession represented by this Academy, is the fact that you here in New York have succeeded in developing a program and holding a meeting such as we have had tonight.

*Milo Helman, D.D.S., F.A.C.D. (New York City):* The relation of diet and of nutrition to dental caries interests me in a general way. What puzzles me is the fact that, in the domain of all living creatures having teeth, the teeth of the human being alone are liable to decay. If nutrition alone is to be blamed for it, why is it that man among all living creatures should encounter such difficulties? Other mammals must eat to sustain life and preserve health. The getting of food is certainly not easier for them than it is for man. Yet, the problem seems to be satisfactorily solved in so far at least as the teeth are concerned, except in the case of man. No dental caries is found in any of the mammals in their wild state. Even when man "tinkers" with the food of mammals, only caries-like manifestations appear. Real tooth-decay has as yet not been definitely demonstrated in lower mammals, although considerable damage has been done to their teeth in experimental feeding.

It appears probable that besides nutrition some additional factors are involved. Microörganisms appear to have a share in this process. But in so far as a complete explanation of cause is concerned it does not quite satisfy. It would seem that casual connections of natural phenomena are at times not so well understood. For instance, is it correct to speak of bacteria alone as causes of disease? While bacteria may be etiologically concerned, do they give us any understanding of the part they play in infection, or of the mechanical connection and nature of the subsequent disease? Does the finding of microörganisms give us the information about their anatomical selection, their mode of origin, their manner of development and the specific expression of anatomical changes that furnish the *objective* basis of disease? Similarly, does the finding of microörganisms in carious cavities prove conclusively that they are the cause of tooth decay? Is there no justification for the assumption that anatomical selection in this instance may furnish the objective basis for tooth decay? If it were not for anatomical selection, all surfaces of a tooth and all teeth in the mouth invaded by the microörganisms would be alike subject to decay. That this is not the case is quite generally known. It may therefore be inferred that tooth decay may not be due to any one cause alone. Thus faulty nutrition alone, microörganisms alone, or anatomical selection alone cannot be blamed for the decay of human teeth. There must be some interplay or combination of all these factors to produce the result.

If there is any direct causal connection, it may be between the objective basis for dental caries and nutrition. From what we have learned from the essayist there is a strong probability, but the proof of it is not so easy to get. Faulty nutrition will no doubt have its effect on the living organism

in general and on some tissues in particular. But in so far as the teeth are concerned, the effect may not be observed for some time. For instance, the effect upon the teeth of faulty nutrition in utero is not seen until long after birth when the deciduous teeth will have erupted; the effect of faulty nutrition upon the teeth during the first year after birth will not be seen until after the eruption of the permanent first molars and incisors; and so on. There is in each of these instances a lapse of years between the time when the causative factor is at work and the time when the effects are observed. In the meantime other disturbances may intervene. What assurance is there to warrant any certainty in the connection of the original cause and the final effect, especially when the individual has not been under continued observation? Even under the most favorable circumstances we are at times puzzled by experiences in actual practice. A specific example will illustrate just what I mean.

A young girl, who has been under the best medical and dental care from birth, presents at the age of fourteen a dentition in superb condition. The teeth are in normal occlusion —the orthodontist took care of that—and with the exception of a few small fillings, inserted by the best dentist in town, the mouth needs no further dental attention. Two or three years pass without the need of dental services. All of a sudden several cavities appear in some of the teeth. We are somewhat surprised; the cavities are filled and the patient is dismissed with the instruction to return in a few months. When the patient returns more cavities are found. We are chagrined. We feel guilty, but don't know why. More fillings, further warnings. This susceptibility to caries goes on for some time and then stops. What has happened? The dentist is puzzled. He prescribed calcium tablets. Did that help? Cod-liver oil would have been better, but girls won't take it, because it makes them fat. The styles of dress you know prescribe a sylph-like figure. Silk hose must cover slender legs. To fit into the modern dress the young lady must not eat too much. Smoking, it is found, appeases hunger and does not add weight. Indeed, girls of twenty weigh less than those of eighteen. Is this situation causally related to the sudden appearance of caries? If so, how? This young lady knows all about orange juice, lettuce, tomatoes, etc. She partakes of them, but does not eat enough for fear she may grow too stout for her clothes.

Do you realize that there are more young people *dieting* than those who know how to choose proper food? Is it not possible that quantity of food, beside quality, may have some effect upon the integrity of tooth substance? Animals do not seem to be particular about the quality, as long as they have enough to eat. Carrion feeders are good examples. Only yesterday I read that the disease-resisting and health-promoting vitamin A can be manufactured by the animal in its body from carotin taken in the food. Carotin as you all know is associated with the yellow pigment found in eggs, butter, carrots and many of the green vegetables we eat.

I would be inclined to propose a reexamination of the entire problem.

So far all inquiries have been made in an *a priori* manner. That is, we are looking for the effect of certain causes. For instance, we establish certain nutritional conditions and expect to find decayed teeth. Suppose we reverse the problem and look for the causes of certain effects. In this connection it would be just as interesting to find out why teeth do *not* decay. Sound teeth seem to be taken for granted, although we have as little knowledge of the reason for sound teeth as we have of the reason for decayed teeth. A thorough investigation of the food habits and state of health of the people who are not afflicted with caries might be of value. It might yield a clue to recommendations of certain measures of which little is known. Such an investigation does not have to be confined to the laboratory. It could be conducted by every dentist in his own office and by the profession, as well as in public schools and public clinics. A collection of reliable data of this sort might throw an entirely different light on the problem now puzzling us. Incidentally similar information might also be obtained from individuals who have carious teeth. Orthodontia was in a similar dilemma. Until very recently every child's dentition appeared to be in need of orthodontic treatment, because the yardstick used to measure it was derived from an imaginary standard. An exhaustive study of the natural development of the face, jaws and teeth furnished the proof that the standard in existence was not known, and that the standard known was not in existence. The practice of orthodontia must now be considerably revised. Many children can now be entirely spared the troublesome procedure, and where it is indicated the treatment can be reduced to a small fraction of the time hitherto thought necessary. What is of interest is that this change in practice came about not through skill and experience in the use of mechanical devices, but through the discovery and recognition of facts, in connection with the development of the face, jaws and teeth, of fundamental importance.

It should therefore be realized that the nutritional expert is concerned with discovering facts of general and fundamental significance. Such facts become of inestimable value in the maintenance of life and elimination of disease. These discoveries are of primary importance and should serve as a biologic background in all work concerned with living beings. But in such specialized fields as ours, it also devolves upon every one concerned to contribute something that will help to solve the problem. The most valuable scientific contribution the individuals composing the dental profession can make is to furnish data from actual experience by which qualified investigators will be enabled to reach definite conclusions, and to indicate practical applications.

*Charles F. Bödecker, D.D.S., F.A.C.D. (New York City):* I do not quite agree with Dr. McCollum or Dr. Mendel in the statement that it is becoming generally believed that diet affects the teeth. I think the idea is still rather skeptically received. McCollum, Mellanby, Percy Howe, and some others, have shown that teeth can be affected by an unbalanced diet, but this refers mostly to teeth in the process of development. Very little work has been done on the effect of diet on teeth already formed. This is the question that deeply concerns human dental decay. In this connection, I believe that prenatal care is less important, as Dr. McCollum very well brought out, than postnatal care of the child. The fetus takes sufficient food, for its bones and tissues, from the bones and tissues of the mother. The deciduous teeth are all developed before birth and are therefore usually well formed. It is only immediately after birth that the permanent teeth begin to be developed and these are dependent on a well-balanced diet for their complete formation.

Dr. McCollum's, as well as Mellanby's, accusation of the cereals is important, because up to a certain time, not long ago, most children were reared on oatmeal or various other cereals with lots of sugar. According to Mellanby, cereals have a particularly evil influence on the teeth. Dr. McCollum mentioned Hanke's experiments on dental decay and periodontoclasia. We know as clinicians that these two diseases lie at opposite extremes: usually if decay is present, periodontoclasia is not; when periodontoclasia sets in, decay usually stops. I do not believe that Hanke's experiments with orange juice will be the open sesame to the causes of dental decay and of periodontoclasia.

Regarding cod-liver oil and the difficulties of administering it, I can add one story about an event in a New York orphanage. The children regularly filed into the room to receive the cod-liver oil from the nurse, opened their mouths, received it, walked out. Later, a fire bucket was discovered outside with a queer greasy-looking fluid. That fire bucket was three-quarters full of cod-liver oil and saliva. The only way to do human experiments properly, or to approach a thorough control, is in orphanages, where there is ample opportunity to control the diet and exercise. Dr. McBeath has worked in two orphanages and made the dental examinations. To our surprise, he found a superb condition of the teeth. The diet had been as close to the optimum as we could have made it. It was a negative result, but one nevertheless which suggests that diet apparently has an influence in stopping decay.

The dental profession undoubtedly is anxious to have some methods of stopping dental decay because they feel the inadequacy of repair work.

This is shown clearly by the reception that Sherman Davis's work received throughout the United States. Medical men received many inquiries from patients whom dentists had told they should consult to have their diet controlled. The physicians wanted to know about the Sherman Davis treatment. The inquiry went to the American Medical Association. You know the result. I am not capable of evaluating the medicaments Sherman Davis used, but he advised also a properly balanced diet—a "shotgun" prescription, which possibly had a good effect. But we have to get nearer to the truth than that in "shotgun" prescriptions.

One point previously brought up is rather disconcerting; namely, people living in the far north, remote from the grocery store, as Dr. Waugh showed in his studies of the Eskimo, have excellent teeth, and they are carnivorous. They live on flesh foods, fish and cod-liver oil, but have no leafy vegetables or fruits. Yet their teeth are in excellent condition. On the other hand, savages in the tropics live on an almost complete vegetarian diet and have good teeth. Therefore, neither vegetarianism nor meat eating seems to be the deciding factor. Jones, Larson, and Pritchard, in Honolulu, advanced the theory that ultraviolet light causes an acid condition of the blood. In the tropics, there is much ultraviolet light in the sunshine. The tropical people eat mainly vegetables and fruits which yield an alkaline ash. Therefore they have acid and alkali in a comparatively proper balance and as a result are in a healthful condition. In the northern climates, on the other hand, the lack of ultraviolet light causes an alkaline condition in the blood. They live on meat which yields an acid ash. Here again there is a proper balance. If that explanation is correct, it has moved out of our way one of the apparent paradoxes in the effect of diet.

When we learn exactly what diet to prescribe for dental health, who is going to prescribe that diet to individual patients? Will the dentist be in a position to do it, or will it be the function of the physician? At present, the average physician is not interested, or is not even keeping up with the modern literature on the subject. When patients ask him which is the proper diet for sound teeth, he evinces very little interest in the matter.

*Leuman M. Waugh, D.D.S., F.A.C.D. (New York City):* I should like to allude briefly to the study I have made of the changing diet of the American Eskimo and the resultant effect upon his teeth. Both in Labrador and in Alaska I found that the old Eskimos who in childhood had lived on the primitive native foods of their ancestors, even though of small stature, had very large well formed and regular jaws, and decay of the teeth was almost absent. It was a common experience to find all of the teeth, without one

carious cavity, present in very many mouths. They were invariably much worn on the biting surfaces, often having been reduced almost to the gum line. The chart projected upon the screen is a tabulation of findings made in Arctic Alaska in the summer of 1929.[2] In the twelve principal native settlements studied, you see that the proteins varied from 25 to 60 percent—five of them being over 40 percent. The fats varied from 25 to 66 percent—eight being over 40 percent. The carbohydrates varied from 15 to 34 percent—four being over 25 percent. The impressive and outstanding fact was that, in proportion as the *children* were fed on the sugars and starches, their teeth were badly decayed. Conversely, in the settlements where they had little or none of these foods they had very little or no dental caries.

The same was true of the studies made in Labrador in the summers of 1924, 1926, and 1927.[3] In the region of Port Burwell, Ungava Bay, of seventy-eight mouths, only six had decayed teeth. From the most reliable sources available, namely the Factor of the Hudson's Bay Post, Mr. Chisley Ford, and from Corporal Nichols of the Royal Canadian Mounted Police, we estimated the diet to average, in protein, 55 to 60 percent; fats, 30 to 35 percent; carbohydrates, 10 percent. Farther south, about the Moravian Missions of Hopedale and Makkovik, the Rev. W. W. Perrett who had been there for over thirty years and in charge of the only stores, estimated that the carbohydrate consumption was about 40 percent. Here the children had most deplorable mouth conditions. Deciduous teeth in children from three to six years of age were decayed and abscessed much worse than among the children of New York. In Alaska I was obliged on three occasions, in three different settlements where white man's foods were available, to extract the abscessed four upper deciduous incisors for tots not three years old. One was only twenty-two months old and not all of the baby teeth had erupted. To me it seems, from observation, that the *metabolism* of carbohydrate food-stuffs may be a factor in this rapid decay. The local influence of carbohydrates seems hardly sufficient to explain the very rapid destruction of the teeth that is taking place among the Eskimos in one or two generations. I feel that a *lowered bodily resistance* to dental caries must at least in part be responsible. This seems to be one of the most important questions before us in the field of *prevention*.

[2] Waugh: *Journal of Dental Research*, 1930, x, p. 387. See also *Ibid.*, 1931, xi, p. 453.
[3] Waugh: *Ibid.*, 1928, viii, p. 428.

# A STUDY OF BLOOD AND SALIVA IN RELATION TO IMMUNITY AND SUSCEPTIBILITY TO DENTAL CARIES[1]

MAXWELL KARSHAN, M.A., PH.D., FRANCES KRASNOW, M.A., PH.D., AND LAURA E. KREJCI, PH.D.

*Department of Biological Chemistry, College of Physicians and Surgeons and School of Dental and Oral Surgery, Columbia University, New York City*

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## I. INTRODUCTION

Little work has been done on the relationship of blood to caries, although saliva has been studied quite extensively. Bödecker's theory of the etiology of dental caries (1), involving the buffering action of the blood as one of the possible factors, and the work of Jones, Larson and Pritchard (14) on dogs, from which they concluded that the decay process may be a clinical indicator of the acid-base balance of the blood, made it desirable to study the alkali reserve and the hydrogen-ion concentration of the blood. Broderick (2) has stated that acidosis is in some way associated with dental decay. Since the possibility existed that dental caries might result from a disturbance in calcium or phosphorus metabolism, we studied these constituents in blood serum. Hawkins (12) has reported that calcium is high and phosphorus is low in individuals with caries.

[1] Conducted with the aid of a grant from the Commonwealth Fund for research in dental caries. Presented at a meeting of the New York Section of the International Association for Dental Research, Dental School of New York University, New York City, May 21, 1931.

As to saliva, the constituents that have been studied most are the neutralizing power, hydrogen-ion concentration, calcium and phosphorus. Marshall (18) attempted to differentiate between immunity and susceptibility to dental caries on the basis of the neutralizing power of saliva, employing the so-called "salivary factor." Shepard and Gies (19) were unable to confirm this. Recently Hawkins (12) reported a higher pH or calcium or both in saliva of persons who are immune from caries than in those who are susceptible. Also Entin and Stark (7) found a higher pH, and Entin (8) reported a higher calcium content, of saliva in immunes than in susceptibles. Bunting and Rickert (3) noted the same for calcium. Horton, Marrack and Price (13) concluded that dental caries is associated with a reduction of the concentration of calcium in saliva. On the other hand Spencer-Payne (20) has been unable to confirm these findings for calcium. As to the phosphorus in saliva, Entin (8) claims higher values for immunes than for susceptibles, although from his figures it is difficult to see how he came to this conclusion. Hawkins reports the reverse. On the basis of a preliminary study, Horton, Marrack and Price (13) found no correlation between phosphorus in saliva and caries.

The present report is a study of the hydrogen-ion concentration, alkali reserve, calcium, phosphorus and protein in blood; hydrogen-ion concentration, titratable alkalinity, calcium and phosphorus in saliva in individuals either immune from or susceptible to dental caries. In addition, a number of individuals, designated "arrested," who had no dental decay for a period ranging from 5 to 15 years immediately preceding were included. The subjects were chosen from the student bodies of Columbia College and the School of Dental and Oral Surgery, Columbia University. The age range of the great majority was 19 to 25 years. About 80 sets of analyses were made involving 17 immune, 6 arrested, and 21 carious individuals. In order to determine the degree of constancy of a particular constituent on different days, more than half of the cases were studied twice; and in several instances tests were made on four different occasions, on the same individual. The intervals between the tests were generally one week.

## II. METHODS

All specimens of blood and saliva were collected before breakast. After obtaining 15 cc. of resting saliva, which usually required about thirty minutes, an additional rest period of fifteen minutes was allowed and then 20 cc. of blood were drawn. This was followed by the collection of approximately 40 cc. of stimulated saliva, which usually took about thirty minutes.

### *Blood*

Blood was drawn (with stasis at start) from an arm vein into a syringe containing about 3 cc. of paraffin oil. The tourniquet was removed immediately after the blood began to flow. About 12 cc. were transferred under oil to a test tube containing dry sodium oxalate. This was covered with paraffin and centrifuged. The plasma was used for the determination of pH, $CO_2$ capacity and $CO_2$ content. The remainder of the blood was used for the preparation of serum for the determination of calcium, inorganic phosphate and protein. The procedures employed were the following:

(a) pH. Hastings and Sendroy (11). Determination was done at room temperature, and the Cullen (5) correction was made.

(b) $CO_2$ capacity and $CO_2$ content. Van Slyke and Neill (22).

(c) Calcium. Kramer and Tisdall method (15), as modified by Clark and Collip (4).

(d) Inorganic phosphorus. Tisdall (21).

(e) Protein. Greenberg (10).

### *Saliva*

Resting saliva was expectorated into a tube, the lower end of which was immersed in paraffin oil contained in a graduated cylinder. Stimulated saliva was collected in the same way, except that paraffin was chewed. Immediately after collection, the pH and titratable alkalinity were determined in both types of saliva, whereas calcium and phosphorus were determined on stimulated saliva only. The procedures employed were the following:

(a) pH was determined colorimetrically on saliva diluted 1:10 using bromthymol blue as indicator. Preliminary tests showed that the

pH does not change as a result of such dilution. Neither was there any change in the pH of the saliva during the period required for collection.

(b) Titratable alkalinity: 3 cc. of saliva were titrated with $N/50$ HCl, using 0.05 cc. methyl orange solution (0.1%) as indicator. Here, too, preliminary tests showed that the titratable alkalinity does not change during the period of collection of the saliva.

(c) Calcium. Leonard's method (17) as modified by the authors (16). Most of the calcium determinations were checked by a method that does not involve ashing and which was developed by the authors during this research (16).

(d) Phosphorus. Tisdall's method (21) for inorganic phosphates in the blood adapted to an HCl solution of ashed saliva. Here too, the analyses were checked by another adaptation of the above method and made applicable to unashed saliva (16).

### III. CHOICE OF CASES

Each individual in the immune group had a full set of teeth, free from cavities and fillings as shown by thorough mouth examination and checked by x-ray. The gum condition was good. All doubtful cases such as those with pits and fissures large enough to admit an explorer were excluded from this group.

In the arrested cases no unfilled cavities were present and the fillings were small in size and few in number, occurred occlusally, and were 5 to 15 years old.

The carious group included those who had active decay associated with definite disintegration of enamel, accompanied by softened dentin. The number of cavities varied from a few to many; some mouths showed involvement in every tooth. In subjects having only a few cavities, the proximal surfaces of the teeth were involved. In these cases teeth other than those with large cavities had areas of decalcification, usually at the gingival margin. Individuals having large cavities, with firm, discolored dentin, were not included.

It is a pleasure to acknowledge our indebtedness to Prof. Daniel E. Ziskin, of the Department of Oral Diagnosis in the Dental School, who made the dental examinations.

## IV. RESULTS

From *Table 1* it is apparent that no differences exist in the alkali reserve of the blood as measured by the $CO_2$ capacity of blood plasma. Minimum and maximum figures and the values between these extremes are about the same in each group.

*Table 3*, giving the pH and $CO_2$ content of blood plasma, likewise fails to show any difference between the various groups. If, as in *fig. 1*, the pH and $CO_2$ content are plotted on the Van Slyke (23) chart as modified by Earle and Cullen (6), it is found that practically all the points fall in the normal area. The distribution of these points is very similar to that reported by Earle and Cullen for apparently normal individuals. *Fig. 2*, in which pH is plotted against $CO_2$ capacity, brings out very strikingly the similarity of distribution in the different groups.

*Table 4* gives the calcium and phosphorus contents in serum. Although the average figures for the immunes and susceptibles are slightly different, being higher for calcium and lower for phosphorus in the caries group, as reported by Hawkins (12), the differences between these average values are so small as perhaps to lack significance. All the values are in the normal range. The results for total serum-protein shown in *Table 2* are all normal and contribute nothing in establishing a difference between immunity and susceptibility.

The results for the pH of both resting (*Table 5*) and stimulated saliva (*Table 6*) indicate higher average values in the immunes than in the susceptibles, as reported by a number of investigators, particularly by Hawkins (12) and by Entin and Stark (7). Our differences are, however, very much less than those reported by the previous authors. Whereas our average pH-values for immunes and susceptibles are respectively 7.03 $\pm$0.15 and 6.90 $\pm$0.12 for resting saliva, and 7.54 $\pm$0.13 and 7.39 $\pm$0.09 for stimulated saliva, Entin reported average pH values of 7.63 for immunes and 7.00 for susceptibles. Most of the pH values in Hawkins' (12) charts fall between 6.6 and 8.1 for immunes and between 6.1 and 7.3 for susceptibles. We do not think that this wide range in pH values, *with so large a number near the upper limit*, represents a true picture of the saliva in the type of individual studied.

Since many investigators have used pH values as a measure of the

TABLE 1

*Data on the carbon dioxide capacity of blood plasma (volume per cent)*

| TYPE OF CASE | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| IMMUNE | | | ARRESTED | CARIOUS | | | | |
| 65.2[1] | 64.8 | 61.7 | 71.2 | 59.5 | 60.1 | 62.6 | 64.8 |
| 65.0 | 63.8 | | 69.0 | 62.8 | 60.8 | 64.0 | |
| 67.3 | | 66.1 | | 62.8 | | | 66.0 |
| | 62.0 | | 62.6 | 61.3 | 64.0 | 61.0 | |
| 60.5 | 59.2 | 63.5 | 68.4 | | 64.0 | | 66.1 |
| 59.7 | | | | 59.1 | | 61.3 | |
| 59.7 | 65.8 | 65.5 | 56.0 | 56.5 | 62.7 | | 66.8 |
| | 61.2 | | | 56.5 | 65.7 | 61.9 | |
| 68.0 | | 66.7 | 59.2 | 58.0 | | | 66.1 |
| 68.7 | 62.8 | | | | 70.0 | 63.8 | |
| | 66.0 | 63.3 | 57.7 | 69.1 | 70.0 | | 70.0 |
| 65.8 | | | | 66.6 | | 60.6 | |
| 64.0 | 67.0 | 64.0 | 59.3 | 66.8 | 65.7 | | |
| | 64.5 | | | | 64.0 | 61.8 | |
| | | 65.0 | | | | | |
| 64.4 ±1.6[2] | | | 61.3 ±5.8[2] | 64.0 ±2.5[2] | | | |

TABLE 2

*Data on the total protein in serum (per cent)*

| TYPE OF CASE | | | | | | | |
|---|---|---|---|---|---|---|---|
| IMMUNE | | | ARRESTED | CARIOUS | | | |
| 6.4[1] | 6.2 | 6.4 | 6.0 | 6.8 | 6.3 | 6.8 | 5.8 |
| 6.1 | 6.6 | | 6.3 | 6.5 | 6.6 | 6.9 | |
| 6.5 | | 6.2 | | 6.1 | | | 6.3 |
| 6.1 | 6.6 | | 6.7 | 6.8 | 6.3 | 6.6 | |
| | 7.0 | 5.9 | 6.2 | | 6.2 | | 6.2 |
| 6.3 | | | | 7.3 | | 6.7 | |
| 5.7 | 7.1 | 6.5 | 6.3 | 6.2 | 6.5 | | 6.4 |
| 6.7 | 7.1 | | | 7.5 | 6.3 | 7.2 | |
| 6.5 | | 6.2 | 5.6 | 7.3 | | | 7.3 |
| | 6.5 | | | | 7.4 | 7.1 | |
| 6.3 | 6.7 | 7.0 | 7.2 | 6.9 | 7.3 | | |
| 6.4 | | | | 6.3 | | 6.0 | |
| | 6.5 | 6.2 | 6.6 | 6.8 | 6.6 | | |
| 6.4 | 6.6 | | | 6.4 | 6.5 | 6.8 | |
| 6.7 | | 6.8 | | | | | |
| 6.5 ±0.2[2] | | | 6.4 ±0.4[2] | 6.6 ±0.3[2] | | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

TABLE 3

*Data on the pH and carbon dioxide content of blood plasma*

| pH | | | | | | | | $CO_2$ CONTENT (VOLUME PER CENT) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Type of case | | | | | | | | Type of case | | | | | |
| Immune | | | Arrested | Carious | | | | Immune | | Arrested | Carious | | |
| 7.43[1] | 7.39 | 7.45 | 7.45 | 7.45 | 7.47 | 7.43 | 7.46 | 68.3 | 69.5 | 71.6 | 65.7 | 70.0 | 67.4 |
| 7.42 | 7.43 | | 7.43 | 7.35 | 7.40 | 7.41 | | 65.7 | 68.5 | 70.8 | 69.1 | 66.0 | |
| 7.41 | | 7.43 | | 7.50 | | | 7.46 | | | | | | 65.9 |
| 7.47 | 7.44 | | 7.45 | 7.43 | 7.46 | 7.44 | | 68.9 | 65.8 | 66.5 | 62.1 | 67.2 | |
| | 7.38 | 7.46 | 7.42 | | 7.42 | 7.42 | 7.41 | 68.5 | | 71.0 | 67.5 | 68.5 | 71.0 |
| 7.47 | | | | 7.31 | | | | | 66.6 | | | | |
| 7.43 | 7.48 | 7.43 | 7.43 | 7.40 | 7.46 | 7.43 | 7.42 | 68.0 | | 61.5 | 68.9 | 68.3 | 73.0 |
| 7.47 | 7.48 | | | 7.42 | 7.45 | 7.41 | | 64.8 | 65.7 | | 71.0 | | |
| 7.43 | | 7.37 | 7.31 | 7.44 | | | 7.51 | | | 63.5 | | 68.7 | |
| | 7.43 | | | | 7.49 | 7.45 | | 70.1 | 66.8 | | 65.1 | | |
| 7.39 | 7.45 | 7.48 | 7.50 | 7.36 | 7.45 | | 7.43 | | | 63.2 | 67.5 | 61.3 | |
| 7.43 | | | | 7.42 | | 7.42 | | 66.6 | 70.0 | | | | |
| | 7.42 | 7.40 | 7.40 | 7.47 | 7.52 | | 7.42 | | | 63.4 | 69.0 | 65.7 | |
| 7.45 | | | | 7.44 | 7.43 | 7.42 | | 66.0 | 67.0 | | 68.5 | | |
| 7.45 | | | | | | | | 67.2 | | | | | |
| 7.43 ±0.02[2] | | | 7.42 ±0.02[2] | 7.44 ±0.02[2] | | | | 67.3 ±1.1[2] | | 65.2 ±2.9[2] | 67.6 ±1.9[2] | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

TABLE 4

*Data on the calcium and phosphorus in serum*

| CALCIUM (MG. PER 100 CC.) | | | | | | | | PHOSPHORUS (MG. PER 100 CC.) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Type of case | | | | | | | | Type of case | | | | | | | |
| Immune | | | Arrested | Carious | | | | Immune | | | Arrested | Carious | | | |
| 10.1[1] | 10.2 | 9.2 | 9.7 | 10.4 | 9.6 | 11.0 | 10.7 | 5.0 | 4.1 | 4.2 | 3.3 | 4.1 | 2.8 | 4.2 | 4.4 |
| 10.5 | 10.0 | | 9.9 | 10.2 | 10.6 | 10.3 | | 4.3 | 3.4 | | 4.3 | 3.0 | 3.2 | | |
| 11.0 | | 11.2 | | 9.8 | | | 11.4 | 4.0 | | 5.4 | | 4.0 | | 3.5 | 4.0 |
| 9.8 | 11.8 | | 9.6 | 10.4 | 11.3 | 9.7 | | 4.1 | 3.9 | | 4.1 | 3.6 | 3.5 | | |
| | 11.1 | 11.0 | | | 10.5 | | 12.5 | | 3.9 | 5.2 | 4.0 | | 3.7 | 3.6 | 3.5 |
| 9.9 | | | 9.6 | 10.4 | | 10.5 | | 5.1 | | | | 4.5 | | | |
| 11.6 | 10.6 | 10.9 | | 9.8 | 10.8 | | 10.5 | 4.7 | 3.9 | 3.1 | 5.0 | 4.0 | 2.7 | 3.2 | 3.5 |
| 10.2 | 10.3 | | 9.9 | 9.8 | 10.6 | 9.9 | | 3.4 | 4.9 | | | 4.2 | 2.7 | | |
| 10.6 | | 11.1 | | 9.9 | | | 11.4 | 4.4 | | 3.5 | 3.8 | 4.1 | | 3.4 | |
| | 10.8 | | 9.6 | | 11.1 | 10.8 | | | 3.3 | | | | 3.3 | | |
| 11.6 | 10.8 | 11.0 | | 11.0 | 10.5 | | | 3.4 | 4.0 | 3.7 | 3.3 | 4.2 | 4.1 | 3.1 | |
| 9.6 | | | 10.4 | 10.2 | | 11.3 | | 3.1 | | | | 4.0 | | | |
| | 10.8 | 10.7 | | 10.2 | 11.5 | | | | 3.8 | 3.8 | 4.4 | 3.6 | 4.0 | 4.5 | |
| 10.2 | | | | | 11.3 | 11.6 | | 4.8 | 3.4 | | | | 3.5 | | |
| 9.5 | 10.4 | | | 10.4 | | | | 3.5 | | 4.4 | | 3.8 | | | |
| | | | | | | | | | | | | 3.6 | 4.7 | | |
| 10.6 ±0.4[2] | | | 9.8 ±0.2[2] | 10.8 ±0.5[2] | | | | 4.1 ±0.5[2] | | | 4.1 ±0.4[2] | 3.7 ±0.4[2] | | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

TABLE 5

*Data on the pH and titratable alkalinity of resting saliva*

| pH | | | | | | | | TITRATABLE ALKALINITY (CC. *N*/50 HCl PER 100 CC.) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Type of case | | | | | | | | Type of case | | | | | | | |
| Immune | | | Arrested | Carious | | | | Immune | | | Arrested | Carious | | | |
| 7.05[1] | 6.65 | 7.15 | 7.20 | 6.85 | 7.05 | 7.05 | 7.15 | 79 | 29 | 80 | 76 | 58 | 56 | 56 | 62 |
| 7.05 | 6.75 | | 7.20 | 6.95 | 7.15 | 6.95 | | 76 | 36 | | 82 | 60 | 53 | 55 | |
| 7.10 | | 7.05 | | 6.85 | 7.05 | | 6.90 | 61 | | 71 | | 65 | 65 | | 44 |
| 7.05 | 6.75 | | 7.05 | 7.05 | | 7.05 | | 60 | 53 | | 74 | 67 | 65 | 43 | |
| | 6.85 | 7.35 | 7.00 | | 7.15 | 6.85 | 6.90 | | 48 | 87 | 54 | | | 48 | 77 |
| 7.15 | | | | 6.85 | 6.85 | | | 61 | | | | 80 | 53 | | |
| 7.05 | 6.85 | 7.15 | 6.95 | 7.10 | 6.85 | 7.15 | 6.95 | 56 | 63 | 119 | 74 | 93 | 58 | 82 | 105 |
| 7.15 | 6.90 | | 7.05 | 7.15 | | 7.05 | | 61 | 71 | | 72 | 82 | 77 | 75 | |
| 7.25 | | 7.05 | | 7.15 | 6.55 | | 6.95 | 56 | | 75 | | 93 | | | 97 |
| | 7.45 | | 7.05 | | 6.55 | 6.95 | | | 74 | | 81 | | 73 | 57 | |
| 6.95 | 7.40 | 7.05 | | 6.65 | | 6.85 | 6.65 | 51 | 78 | 59 | | 57 | 59 | 50 | 78 |
| 6.75 | | | 7.05 | 6.95 | 7.00 | | | 51 | | | 53 | 54 | | | |
| | 6.95 | 7.15 | | 6.90 | 6.95 | 6.70 | 6.55 | | 55 | 54 | | 56 | 32 | 71 | 67 |
| 6.85 | 7.10 | | 7.15 | 6.95 | | 6.75 | | 52 | 58 | | 56 | 68 | 41 | 69 | |
| 6.75 | | 6.75 | | | 6.85 | | 6.90 | 64 | | 70 | | | | | 57 |
| 7.03 ±0.15[2] | | | 7.08 ±0.06[2] | 6.90 ±0.12[2] | | | | 67 ±9[2] | | | 68 ±9[2] | 67 ±9[2] | | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

TABLE 6

*Data on the pH and titratable alkalinity of stimulated saliva*

| pH | | | | | | | | | TITRATABLE ALKALINITY (CC. *N*/50 HCl PER 100 CC.) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Type of case | | | | | | | | | Type of case | | | | | | | | |
| Immune | | | Arrested | Carious | | | | | Immune | | | Arrested | Carious | | | | |
| 7.45[1] | 7.25 | 7.35 | 7.50 | 7.25 | 7.25 | 7.35 | 7.40 | 7.45 | 114 | 89 | 87 | 114 | 91 | 112 | 100 | 115 | 87 |
| 7.45 | 7.35 | | 7.45 | 7.35 | 7.25 | 7.35 | 7.30 | | 143 | 100 | | 105 | 97 | 117 | 98 | 111 | |
| 7.35 | | 7.45 | | 7.25 | 7.40 | | | 7.15 | 107 | | 151 | | 105 | 116 | | | 98 |
| 7.35 | 7.30 | | 7.35 | 7.35 | 7.35 | 7.50 | 7.50 | | 105 | 103 | | 131 | 96 | 106 | 97 | 152 | |
| | 7.30 | 7.85 | 7.35 | | | 7.25 | | 7.35 | | 107 | 158 | 121 | | | 100 | | |
| 7.55 | | | | 7.60 | 7.45 | | 7.25 | | 132 | | | | 152 | 126 | | 81 | |
| 7.45 | 7.60 | 7.50 | 7.25 | 7.45 | 7.55 | 7.35 | | | 125 | 150 | 146 | 97 | 160 | 148 | 91 | | |
| 7.45 | 7.75 | | 7.25 | 7.55 | 7.35 | 7.55 | 7.35 | | 123 | 143 | | 85 | 153 | 148 | 96 | 113 | |
| 7.65 | | 7.50 | | 7.60 | | | | | 130 | | 134 | | 136 | | | | |
| | 7.55 | | 7.45 | | 7.25 | 7.60 | 7.40 | | | 108 | | 134 | | 77 | 180 | 131 | |
| 7.55 | 7.45 | 7.55 | | 7.45 | 7.35 | 7.75 | | | 153 | 112 | 125 | | 143 | 92 | 171 | | |
| 7.45 | | | 7.35 | 7.45 | | | 7.25 | | 141 | | | 89 | 122 | | | 102 | |
| | 7.45 | 7.45 | | 7.50 | 7.45 | 7.35 | | | | 128 | 99 | | 125 | 152 | 109 | | |
| | 7.40 | | | 7.55 | 7.45 | 7.45 | | | 92 | 119 | | | 142 | 134 | 115 | | |
| 7.35 | | | 7.55 | | | | | | 88 | | 189 | 120 | | | | 103 | |
| 7.25 | | 7.50 | | | | | | | | | | | | | | | |
| 7.54 ±0.13[2] | | | 7.40 ±0.09[2] | 7.39 ±0.09[2] | | | | | 126 ±22[2] | | | 118 ±22[2] | 116 ±21[2] | | | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

TABLE 7

*Data on the calcium and phosphorus of stimulated saliva*

| CALCIUM (MG. PER 100 CC.) | | | | | | | | | PHOSPHORUS (MG. PER 100 CC.) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Type of case | | | | | | | | | Type of case | | | | | | | |
| Immune | | | Arrested | Carious | | | | | Immune | | | Arrested | Carious | | | |
| 4.7[1] | 5.3 | 4.6 | 4.7 | 3.5 | 5.3 | 6.1 | 5.2 | 6.3 | 12.0 | 10.5 | 9.9 | 8.8 | 10.6 | 10.9 | 10.2 | 7.5 |
| 5.6 | 5.2 | | 4.9 | 4.2 | 4.4 | 6.6 | 5.4 | | 11.7 | 10.1 | | 9.3 | 10.8 | 11.4 | 10.1 | |
| 5.5 | | 5.7 | | 3.8 | 5.0 | | | 5.9 | 10.6 | | 10.8 | | 10.0 | 9.7 | | 8.8 |
| 5.1 | 5.0 | | 4.7 | 4.2 | 5.2 | 6.4 | 6.0 | | 10.4 | 9.9 | | 12.5 | 10.4 | 9.0 | 8.7 | |
| | 5.1 | 6.9 | 4.0 | | | 6.5 | | 5.9 | | 9.6 | 11.7 | 13.4 | | | 8.9 | 10.3 |
| 4.4 | | | | 5.4 | 4.7 | | 4.7 | | 8.8 | | | | 11.1 | 9.4 | | |
| 4.6 | 5.5 | 5.4 | 8.7 | 4.2 | 4.9 | 5.1 | | | 9.6 | 14.0 | 15.4 | 13.6 | 10.5 | 9.9 | 7.6 | 11.7 |
| 4.5 | 5.5 | | 8.1 | 4.8 | 5.4 | 5.0 | 5.2 | | 10.4 | 14.0 | | 12.6 | 10.8 | 9.3 | 6.7 | |
| 4.7 | | 7.8 | | 4.8 | | | | | 10.0 | | 12.9 | | 8.8 | | | 8.0 |
| | 5.5 | | 4.5 | | 4.1 | 5.2 | 5.4 | | | 9.4 | | 11.4 | | 10.1 | 12.6 | |
| 5.8 | 5.6 | 7.0 | | 4.7 | 3.9 | 4.8 | | | 8.9 | 10.0 | 13.2 | | 8.7 | 10.8 | 12.5 | 10.8 |
| 6.1 | | | 4.1 | 4.2 | | | 5.8 | | 9.3 | | | 13.2 | 10.2 | | | |
| | 7.1 | 7.6 | | 4.3 | 5.5 | 5.3 | | | | 13.1 | 9.8 | | 8.5 | 8.5 | 8.7 | 13.1 |
| 4.7 | 9.2 | | 5.6 | 4.4 | 5.7 | 6.1 | | | 10.6 | 14.5 | | 9.9 | 9.8 | 9.0 | 8.4 | |
| 4.9 | | 6.0 | | | | | | | 10.0 | | 10.1 | | | | | 10.1 |
| | 5.6 | | | | | | | | | | | | | | 10.9 | |
| 5.9 ±0.9[2] | | | 5.3 ±1.1[2] | 5.3 ±0.6[2] | | | | | 11.3 ±1.6[2] | | | 11.6 ±1.6[2] | 9.9 ±1.2[2] | | | |

[1] In all tables figures grouped in this way represent results obtained on the same individual at different times.

[2] An average was obtained for each group of figures, and these averages were averaged. These are given with the average deviation.

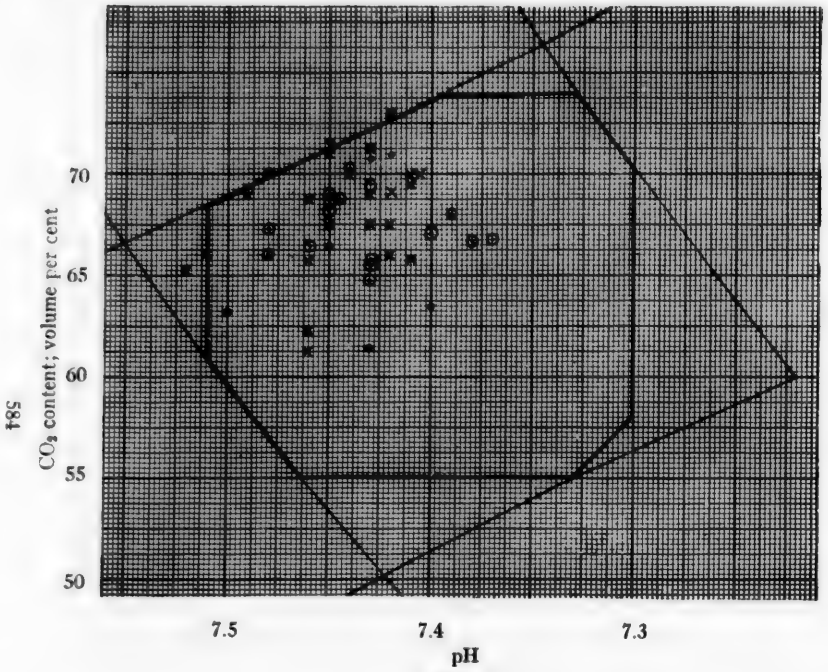


FIG. 1

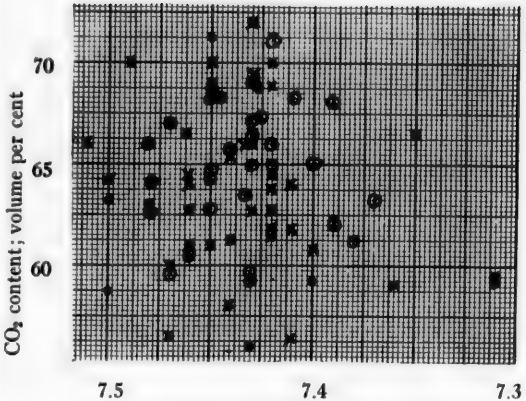


FIG. 2

In each figure ⊙ = immune; ● = arrested; × = susceptible

FIG. 1. THE RELATION OF pH TO CARBON DIOXIDE CONTENT OF BLOOD PLASMA, SHOWING THE SIMILARITY OF DISTRIBUTION OF THE POINTS REPRESENTING THE VARIOUS TYPES OF CASES

FIG. 2. THE RELATION OF pH TO CARBON DIOXIDE CAPACITY OF BLOOD PLASMA, SHOWING THE SIMILARITY OF DISTRIBUTION OF THE POINTS REPRESENTING THE VARIOUS TYPES OF CASES

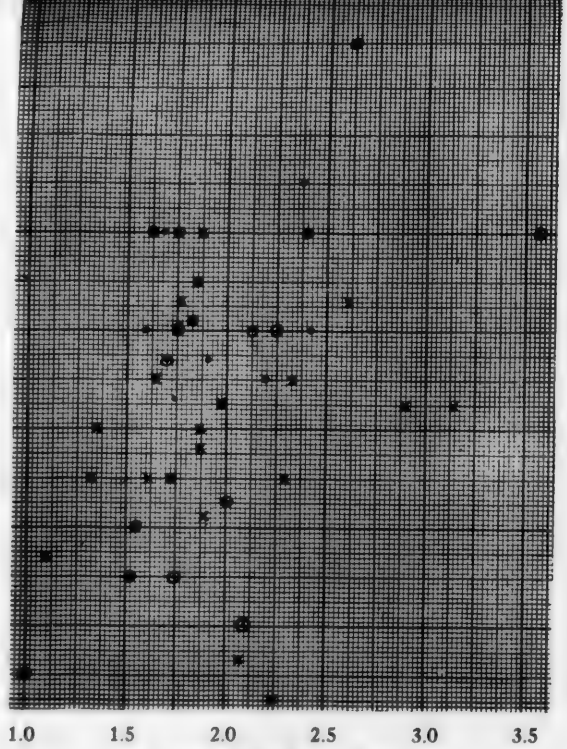


FIG. 3

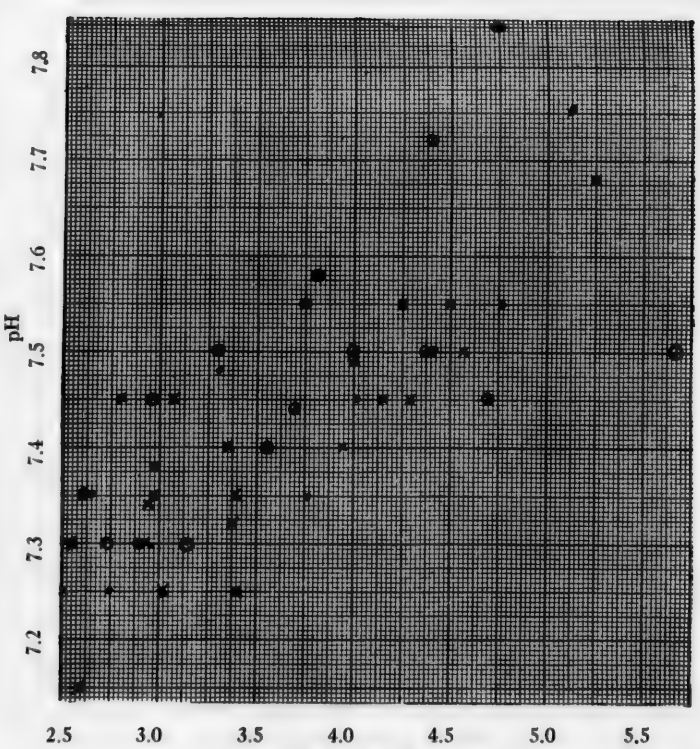


FIG. 4

In each figure ⊙ = immune; ● = arrested; × = susceptible

FIG. 3. THE RELATION OF pH AND TITRATABLE ALKALINITY OF RESTING SALIVA, SHOWING A LACK OF STRICT PROPORTIONALITY BETWEEN THESE TWO VALUES.

That a considerable degree of correlation exists is shown by the absence of points in the upper left and lower right areas. Two values of 2 cc. of HCl for two "susceptible" salivas at pH 6.5 were not included in the figure.

FIG. 4. THE RELATION OF pH AND TITRATABLE ALKALINITY OF STIMULATED SALIVA, SHOWING THE LACK OF STRICT PROPORTIONALITY BETWEEN THESE VALUES.

That a considerable degree of correlation exists is shown by the absence of points in the upper left and lower right areas.

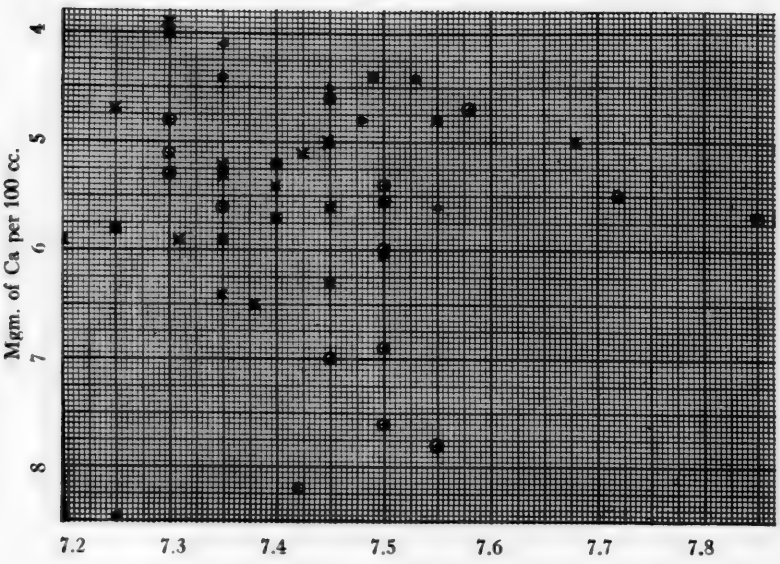


FIG. 5

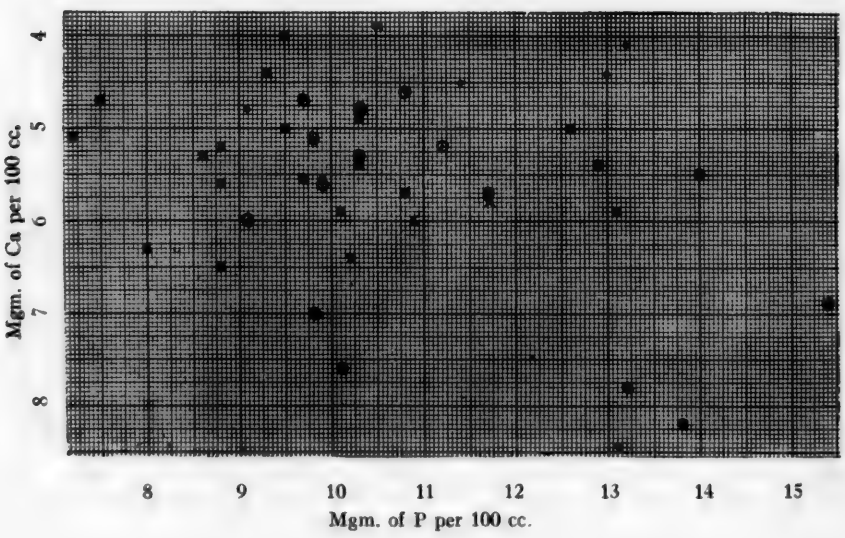


FIG. 6

In each figure ⊙ = immune; ● = arrested; × = susceptible

FIG. 5. THE RELATION OF pH AND CALCIUM OF STIMULATED SALIVA

With the exception of a number of high-calcium values from the immune group, there is a great similarity in the distribution of the points in the various groups.

FIG. 6. THE RELATION OF CALCIUM AND PHOSPHORUS IN STIMULATED SALIVA

A fairly large proportion of the immune cases are found in the right section representing high-phosphorus values. On the other hand, the left section, indicating low-phosphorus values, contains many points representing individuals in the carious group.

buffering or neutralizing power of saliva, we compared the pH of each specimen of saliva with the titratable alkalinity (*Tables 5* and *6*), using methyl orange as indicator, as was done by Gies and Whiteford (9) who demonstrated the value of this indicator for this purpose. Titratable alkalinity with this indicator is probably a better criterion, than the pH, of the neutralizing power of saliva. If the pH is plotted against titratable alkalinity (*figs. 3* and *4*), the points are scattered instead of falling on a definite line, as would be the case if pH and titratable alkalinity were proportional. However, a considerable degree of correlation is indicated by the absence of points in the upper left and lower right areas. The average values for titratable alkalinity of stimulated saliva show the same general difference between immunes and susceptibles as the pH values; but the values for titratable alkalinity of resting saliva are alike for immunes and susceptibles.

The average calcium values (*Table 7*) are higher in immunes than in susceptibles as reported in the literature, but here too the difference is not so great as some others have reported. Entin (8) recorded calcium values ranging from 5 to 33 mg. per 100 cc. of saliva, with an average of 16.3 for immunes and 14.0 for susceptibles. We are at a loss to understand how these high values were obtained, unless these workers titrated the suspended matter in saliva along with the calcium, precipitated as calcium oxalate. By making the determination for calcium in this incorrect way, we have been able to obtain abnormally high calcium values such as have been obtained by these authors. Also, we cannot see how Hawkins (12) obtained so many calcium values below 3.5 mg. per 100 cc. of saliva. In *fig. 5* we have plotted pH against calcium in a manner similar to Hawkins (12). Although a number of immunes give points corresponding to high calcium values, most of the values overlap.

The average phosphorus values (*Table 7*) are somewhat greater in the immunes than in the susceptibles. As shown in *fig. 6*, in which phosphorus is plotted against calcium, a fairly large proportion of points representing the immunes are grouped in the upper range, whereas a large proportion representing susceptibles are grouped in the lower range.

## V. CONCLUSIONS

This report is a study of the hydrogen-ion concentration, alkali reserve, calcium, phosphorus and protein, in blood; hydrogen-ion concentration, titratable alkalinity, calcium and phosphorus in saliva in individuals either immune from or susceptible to dental caries. Some of the constituents show average values which differ somewhat for the different groups. However, these differences are slight compared with those obtained by other workers and may lack significance in view of the overlapping of so many of the individual values. The main difference was found in the phosphorus content of saliva, this having been higher in many instances in immune persons than in those susceptible to caries.

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# DENTAL CARIES FROM A BIOCHEMICAL STANDPOINT[1]

J. C. FORBES, PH.D.

*Chemical Department, Medical College of Virginia, Richmond, Virginia*

The etiology of dental caries has been discussed by many investigators in recent years, but there has been no agreement regarding it. Many have claimed that the presence of acid-forming bacteria in the mouth is the all important factor; others see the reaction of the saliva as the decisive one. The calcium content of the saliva has been considered a factor—low content of calcium indicating susceptibility; high content, immunity. The presence or absence of certain vitamins in the diet has been regarded as a controlling condition, while others consider that the potential alkalinity or acidity of the diet is the important thing, a potentially-acid diet favoring caries and an alkaline one giving immunity. This paper has consequently been written, not with the idea of presenting new facts but in an attempt, from a chemical standpoint, to correlate the available data, which show that all of these may be factors in the production of the disease. They must be considered not as independent factors but in relationship to one another, and as each and all influence the teeth.

Since dental caries is, at the beginning, primarily a solution of the enamel substance, we can get some idea of the mechanism involved if we simply examine what happens when any insoluble substance goes into solution. If, for example, calcium phosphate is placed in water, the following condition of equilibrium is established:

$$Ca_3(PO_4)_2 \rightleftarrows Ca_3(PO_4)_2 \rightleftarrows 3\ Ca^{++} + 2\ PO_4^{---}$$

Solid Solution Ions

This is a case of dynamic equilibrium, under which, when established, the following condition holds true: $(Ca^{++})^3 \times (PO_4^{---})^2 = k$ (ioniza-

[1] Read before a meeting of the Richmond Dental Society, Richmond, Va., January 22, 1931.

tion constant); that is to say, the product of the calcium-ion concentration cubed, times the phosphate-ion concentration squared, is equal to a constant (ionization constant). This constant varies with the temperature, but is not influenced by the relative concentration of the calcium and phosphate ions. If, for example, the calcium-ion concentration of the solution is increased by the addition of a soluble calcium salt, like calcium chloride, the product of the calcium-ion concentration cubed, times the phosphate-ion concentration squared, will be momentarily greater than $k$; but combination of the calcium and phosphate ions will then take place, forming calcium phosphate, until once more the product $(Ca^{++})^3 \times (PO_4^{---})^2$ becomes equal to $k$. The final result is an increased calcium-ion concentration over the initial value, but a decreased phosphate-ion concentration. On the other hand, if the concentration of the Ca and $PO_4$ ions is diminished in any way, the product of the $(Ca^{++})^3 \times (PO_4^{---})^2$ will be momentarily less than $k$, as some of the $Ca_3(PO_4)_2$ molecules in solution will ionize, and an equivalent amount of the solid $Ca_3(PO_4)_2$ passes into solution. This continues until once more the product of the $(Ca^{++})^3 \times (PO_4^{---})^2 = k$. If the reaction diminishing the Ca- or $PO_4$-ion concentration continues, then finally all of the solid $Ca_3(PO_4)_2$ passes into solution. This is well exemplified by the action of an acid, like hydrochloric acid, on $Ca_3(PO_4)_2$. It has already been mentioned that when $Ca_3(PO_4)_2$ is placed in water the following equilibrium is established:

$$\underset{\text{Solid}}{Ca_3(PO_4)_2} \rightleftarrows \underset{\text{Solution}}{Ca_3(PO_4)_2} \rightleftarrows \underset{\text{Ions}}{3\ Ca^{++} + 2\ PO_4^{---}}$$

If to this solution HCl is added, the H ions from the HCl immediately react with the $PO_4$ ions forming $HPO_4$ ions, thus diminishing the $PO_4$-ion concentration. This diminution in $PO_4$-ion concentration causes some of the $Ca_3(PO_4)_2$ molecules in solution to ionize, an equivalent amount of the solid $Ca_3(PO_4)_2$ passing into solution in an attempt to increase once more the product of the $(Ca^{++})^3 \times (PO_4^{---})^2$ to $k$. When sufficient acid is added, all of the $Ca_3(PO_4)_2$ is finally changed into $CaHPO_4$, which by the further addition of acids may be changed into the soluble $Ca(H_2PO_4)_2$.

Since the enamel is primarily composed of inorganic salts we must assume that its solution follows the same lines as that of any other insoluble inorganic salt. Also, since the enamel is primarily composed of calcium and phosphates, we may assume for this discussion that it consists mainly of $Ca_3(PO_4)_2$, feeling that what holds true for this salt must hold true, with limitations, for the substances of which enamel is composed. We have seen that the solution of $Ca_3(PO_4)_2$ is dependent on the concentration of the Ca and $PO_4$ ions. If the product of $(Ca^{++})^3 \times (PO_4^{---})^2$ is momentarily increased over the value $k$ (ionization constant), deposition or precipitation of $Ca_3(PO_4)_2$ takes place; but if the product is reduced below $k$, solution of the $Ca_3(PO_4)_2$ proceeds until the product is again increased to $k$. It would therefore seem that those factors which influence the concentration of Ca and $PO_4$ ions, in the saliva bathing the teeth in any particular locality, must be determining factors in the etiology of caries.

*Factors influencing the $PO_4$-ion concentration.* The chief factors involved in determining the $PO_4$-ion concentration of the solution bathing the teeth in any locality are likely to be (a) the phosphate concentration and (b) the H-ion concentration of the solution in that locality. The influence of the former is evident and requires no further comment, but a discussion of the latter may be in order. It was shown when considering the solution of $Ca_3(PO_4)_2$ in acids that the first step in the reaction was a reduction of the $PO_4$-ion concentration through a combination of the $PO_4$ ions with the H ions to form $HPO_4$ ions. This may be readily seen when one considers the equilibrium equation representing the reaction $H^+ + PO_4^{---} \rightleftarrows HPO_4^{--}$ Since this is a state of dynamic equilibrium, it follows from the law of mass action that any increase in the concentration of either of the ions on the left-hand side of the equation causes the equilibrium to shift to the right, with the formation of additional $HPO_4$ ions. For example, if the H-ion concentration is increased by the addition of acid, some of the H ions combine with the $PO_4$ ions to form $HPO_4$ ions, and a corresponding decrease in the concentration of the $PO_4$ ions ensues. In a similar way an increase in the $PO_4$-ion concentration causes a decrease in the H-ion concentration.

The H-ion concentration of the solution bathing the teeth in any place is determined (a) by the neutralizing action of the saliva, (b)

the amount of acid formed at any particular place, (c) the accessibility of the saliva, and (d) the carbonic-acid tension of the solution. The first three are likely the primary factors. In the absence of acid production in the mouth, the H-ion concentration is determined by that of the secreted saliva and the carbon-dioxide tension of the solution; but in the presence of acid production, it is determined primarily by the amount of acid formed and the ability of the saliva to neutralize it. The latter is determined, of course, by the neutralizing power and accessibility of saliva. If the acid production, for example, takes place under a plaque where saliva has no free access, the same amount of acid causes a greater H-ion concentration than when the same amount of acid is produced on a free surface well exposed to the saliva, for the latter neutralizes the acid and washes it away. The amount of acid produced is determined by various factors, such as the organisms present and the amount of available carbohydrate, since these acids come primarily, if not altogether, from carbohydrate fermentation.

It should be noted that the neutralizing action of the saliva is not necessarily proportional to the H-ion concentration, i.e., pH, of the saliva as is generally believed. If, for example, we consider water of pH 7, and a buffer solution of pH 6, we can readily see that the former has practically no neutralizing action, while the latter, in spite of the fact that it itself is slightly acid to true neutrality, pH 7, may have a very marked neutralizing action against acids such as lactic acid. If the materials composing two buffer solutions are the same, and their pH is the same, their neutralizing power will be proportional to the amount of buffer present. The neutralizing action of the saliva against acids depends upon not only the pH, but also the amount of buffers present. A high pH and a high concentration of buffer salts, such as phosphates, indicate a marked neutralizing action; a high pH and a low concentration of buffer salts, a moderate neutralizing action; a low pH and a high concentration of buffers, a moderate neutralizing action; and a low pH and a low concentration of buffer salts, a low neutralizing action. One would expect caries to be rampant under the last condition, to occur occasionally under the second and third conditions and to be absent under the first. It is therefore possible that some of the discrepancies in the theory that a low pH of

the saliva indicates caries, and a high pH indicates immunity, may be due to variations in the neutralizing power of the various salivas.

*Influence of calcium concentration.* When we consider the equilibrium

$$\underset{\text{Solid}}{Ca_3(PO_4)_2} \rightleftarrows \underset{\text{Solution}}{Ca_3(PO_4)_2} \rightleftarrows \underset{\text{Ions}}{3\ Ca^{++} + 2\ PO_4^{---}}$$

we see that the concentration of the calcium ions is just as important as that of the $PO_4$ ions. If the Ca-ion concentration is high, then, in order that solution of the enamel may take place, the $PO_4$-ion concentration must be lower than when the calcium concentration is low. This is in agreement with Hawkins' (1) results. He has shown that when the salivary calcium is high the reaction of the medium may be more acid without any signs of caries than when the salivary calcium is low. In other words, it will require a greater acid concentration, in this case a lower $PO_4$-ion concentration, to cause solution of enamel when the calcium concentration is high than when it is low.

*Factors influencing salivary pH, calcium and phosphorus.* According to Starr (2) the salivary pH is determined by the $BHCO_3/H_2CO_3$ ratio of that secretion, and the concentration of these in the saliva is likely determined by that of the blood. When the $BHCO_3$ concentration of the blood is high, and the $H_2CO_3$ low, the saliva has a high pH, while a high $H_2CO_3$ content of the blood, and consequently of the alveolar air, results in an acid saliva; that is, saliva having a low pH. Concerning the calcium and phosphorus concentrations, the work done is insufficient to give us much information on the factors influencing them. It is likely that the amount in the food is an important factor, and also the general metabolic condition of the individual.

*Arrested caries.* If we are to assume that caries is altogether due to the influence of external forces on the teeth, how shall we explain the condition in arrested caries? According to Broderick (3), arrested caries is never found in pulpless teeth, indicating that a definite response of the living tooth itself is necessary in addition to a suitable environment. There is also a condition of hypercalcification under the cavity, indicating that the tooth is reacting to a stimulus

and is trying to overcome the carious process. It is likely that in all cases of caries in vital teeth, there is a similar reaction and the final result is determined by the strength of the opposing forces. If the calcifying factors are the strongest, the caries is arrested; but if the decalcifying factors are in the ascendency, the caries progresses. Also cases are known in which decay has been rampant in vital teeth, but pulpless teeth in the same individual have remained free from caries. It is now generally accepted that the dentine is in direct communication with the body fluids, and a lot of evidence in recent years indicates that the same is also true of the enamel. It is well known that a prolonged period of acidosis causes marked decalcification of the bony structures and teeth. Pritchard (4) et al. report that in young children teeth tend to decay during the period of rapid bone-formation, indicating that calcium may be removed from the teeth to supply material for the more important structures. The mechanism of decalcification is not well understood, but it is favored by a prolonged acidosis. Inversely, the feeding of a potentially-alkaline diet favors calcium and phosphorus retention (Cole et al. —5). It is important to distinguish here between a diet that is *potentially* alkaline and one that is *actually* alkaline. The latter may cause diminished calcium retention owing to decreased calcium absorption from the intestines on account of the ensuing marked alkalinity of the intestinal content; the former does not change the intestinal reaction, yielding alkaline salts only when oxidized in the body.

It would therefore seem that in a case of arrested caries conditions somewhat like the following might apply. An acute illness might have produced a marked condition of acidosis, with a corresponding increase in the acidity of the saliva, and possibly a decrease in the calcium and phosphorus concentrations, owing to increased elimination through the kidneys and, likely, a diminished intake. Such a condition would favor solution of the enamel and, in conjunction with acid-forming bacteria in the mouth, might result in the formation of a definite carious condition. If the individual recovered, the food and salt consumption increased; the acidosis disappeared, or was changed to a slight alkalosis; the pH and neutralizing action of the saliva probably increased. These conditions, together with decrease in acid-forming bacteria as noted by Bunting (6) through feeding a well

reinforced diet, would favor calcification and arrest of the carious process.

*Discussion.* It would seem that the determining factors in dental caries are primarily the acid-base equilibrium of the blood on the one hand, and on the other the Ca- and $PO_4$-ion concentrations of the solution in contact with the teeth in any particular locality. The Ca-ion concentration is primarily determined by that of the saliva. The most important factor affecting the $PO_4$-ion concentration is the H-ion concentration of the solution at any particular place, a high H-ion concentration producing a low $PO_4$-ion concentration, owing to a combination with the $PO_4$ ions to give $HPO_4$ ions and a consequent diminution in the concentration of the $PO_4$ ions. We thus see that the neutralizing power of the saliva against acids becomes a very important factor in the prevention of caries. It is likely that this is primarily determined by the alkali reserve of the blood. Although Starr has shown that the pH of the saliva is directly proportional to the $CO_2$ content of the alveolar air and not to the blood alkali reserve, it is however likely that its neutralizing action against acids closely follows the alkali reserve. We can therefore readily see how a potentially-alkaline diet, by increasing the blood alkali-reserve, prevents caries, as Pritchard has shown, while a potentially-acid diet favors decay. Pritchard reports from his investigations that in Hawaii he has seen no cases of caries in children over three years of age who lived on a potentially-alkaline diet. Similarly, Bunting (6) has shown that diets high in vegetables and milk practically eliminate caries in children. He does not state whether the diets as a whole would be potentially acid or alkaline, but judging from the fact that the children received a quart of milk a day and a high-vegetable diet they were presumably potentially alkaline, since both of these dietary factors are potentially alkaline in reaction.

A condition of acidosis that favors decalcification of the bony structures and teeth from within evidently also favors the destruction of the teeth from without by the production of an acid saliva, with a consequently low neutralizing action on acids. Similarly, a potentially-alkaline diet, tending to keep the alkaline reserve towards a high normal, promotes calcification, and also favors prevention of tooth decay from without owing to the greater neutralizing action of the saliva and possibly to an effect on the oral flora.

The foregoing is by no means a complete explanation of the phenomena of caries. It is presented only to show that all of the factors, such as calcium concentration, phosphate concentration, H-ion concentration, neutralizing power of the saliva against acids, acid-base equilibrium of the blood, etc., must be considered not as independent factors but in relationship to one another and as each and all influence the solution of the enamel.

It is a pleasure to acknowledge the helpful criticism of Dr. Harry Bear, Dean of the School of Dentistry, in the preparation of this paper.

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# DENTAL ORIGIN OF REFLEX FACIAL PAIN[1]

LESTER RICHARD CAHN, D.D.S.

*Department of Oral Pathology, Dental School, Columbia University*

I have never seen a case of true trigeminal neuralgia that was cured by the extraction of a tooth. On the other hand, I believe that the largest percentage of minor facial neuralgias and otalgias, where no disease is present in the ear, is of dental origin. The most important factor in the causation of neuralgia of the face and head is disease of the dental pulp. Because of its importance it is well to briefly review the neuro-anatomy and pathology of this tissue.

The dental pulp is that mass of soft tissue occupying the center of the crown of the tooth or pulp chamber, and the canals of the roots. It is composed of a loosely woven connective tissue, whose cells are of mesenchyme character (*fig. 1*), capillaries and nerves. At its periphery, adjacent to the dentine, are columnar cells called the odontoblasts. Processes known as Tomes' fibres are prolongations of the odontoblasts that enter the tubules of the dentine (*fig. 2*). The nerves of the pulp are abundant (*fig. 3*). In proportion to its size it is more highly innervated than any organ in the body. In the upper jaw the pulpal nerves are branches of the posterior, middle, and anterior dental nerves, which in turn are given off by the superior maxillary division of the fifth nerve. In the lower jaw the nerves of the pulp are derived from the inferior dental nerve, a branch of the third division of the trigeminal. The cells of the pulpal neurites lie in the gasserian ganglion. Fasciculi of medulated nerve fibres enter the apical foramen of the root, divide into numerous smaller bundles and run toward the crown, ending in plexuses beneath the odontoblastic layer. Nerve endings have been demonstrated between the odontoblasts (*fig. 4*). Whether the dentine is innervated or not, has never been satisfactorily proven. Clinically the dentine is often extremely sensitive. The

[1] Read at a meeting of the American Stomatological Association, Columbia University Club, New York City, February 17, 1931.

FIG. 1. GROUND SUBSTANCE OF PULP
Note the mesenchyme character of the cells

FIG. 2. DENTAL PULP

*A*, odontoblasts. *B*, Tomes' fibers; pulled out from dentinal tubules. *C*, nerve fiber entering odontoblastic layer.

fibres of Tomes may in some way, not yet made clear, be responsible for the transmission of sensation from the dentine to the pulp.

As for the pathology of the pulp, this organ undergoes, in general, the same inflammatory and degenerative changes as do all the other

FIG. 3. NERVE FASCICULI IN DENTAL PULP

It is astonishing to observe so many nerve bundles in an area so limited. One can easily realize, from looking at this picture, that any inflammation or calcification is bound to involve these nerves.

tissues. Ordinarily the pulp should be a well protected tissue. It is encased in the hard structures of the tooth and appears to be safely guarded against outside influences. Unfortunately this is not the

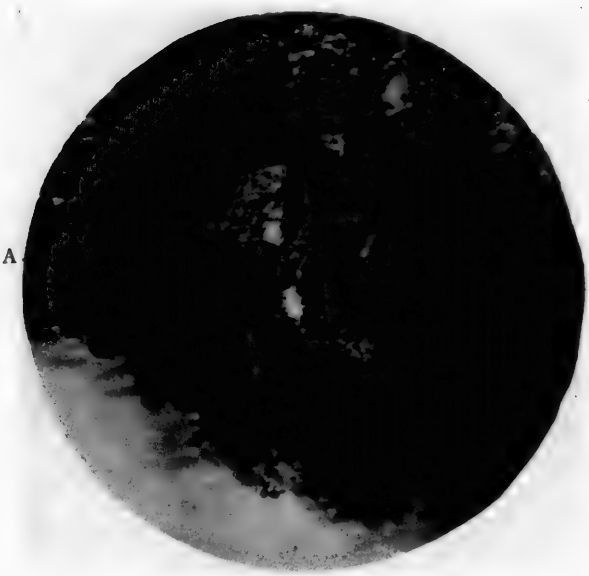


FIG. 4. DENTAL PULP
*A*, nerve ending in odontoblasts

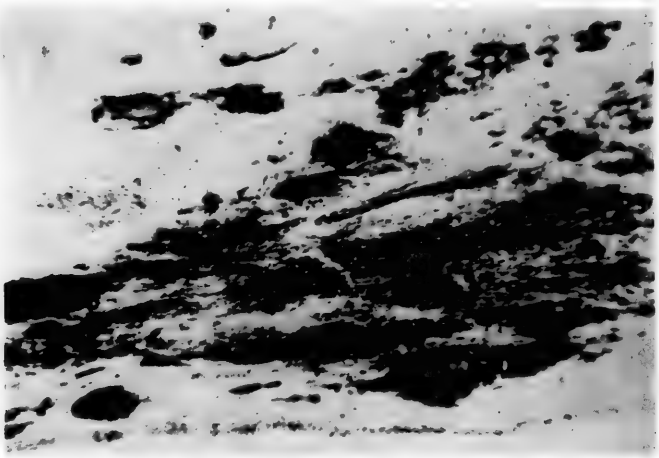

FIG. 5. DIFFUSE CALCIFICATION IN PULP

case. Dental caries is a prevalent disease, and the enamel of the tooth is quickly destroyed and penetrated. At the instant of exposure of the dentine, pulp involvement is imminent because bacteria and their products can reach this tissue via Tomes' fibres and the dentinal tubules. Pulp contamination also arises from continuity of infection along the periodontal membrane, as in cases of suppurative periodontoclasia (pyorrhea alveolaris). Thus pulp disease is one of the commonest ailments.

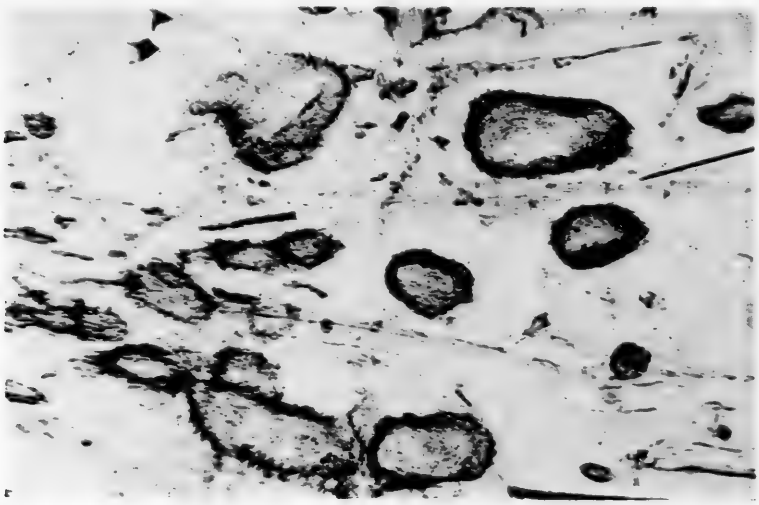

FIG. 6. EXTENSIVE CALCIFICATION IN PULP OF PYORRHETIC TOOTH THAT HAD NEVER BEEN AFFECTED BY CARIES

*A*, nerve trunk passing between two islands of calcification

In the histo-pathology of the dental pulp the most frequently encountered lesion is calcific infiltration. We are all familiar with the term "pulp stone." This phrase has been used to describe generically all types of pulpal calcification; however we have found that calcific change takes two forms. One is composed of dentine, and represents a perverted growth of that tissue. The other is a true deposition of calcium salts. The real cause of the abnormal dentinal calcification is unknown. True calcific infiltration is always associated with some previously diseased condition and sluggish circulation. Fatty

and hyaline degenerations frequently precede it. This type also assumes two forms: one, in which the salts are deposited in a circumscribed area as though a certain spot was hardened in order to obliterate it; the other, a diffuse infiltration of what appears to have been fibrous tissue (*fig. 5*). A tooth harboring a pulp having calcific deposits need never have been affected by dental caries. I have found calcifications in embedded upper cuspids that never were decayed;

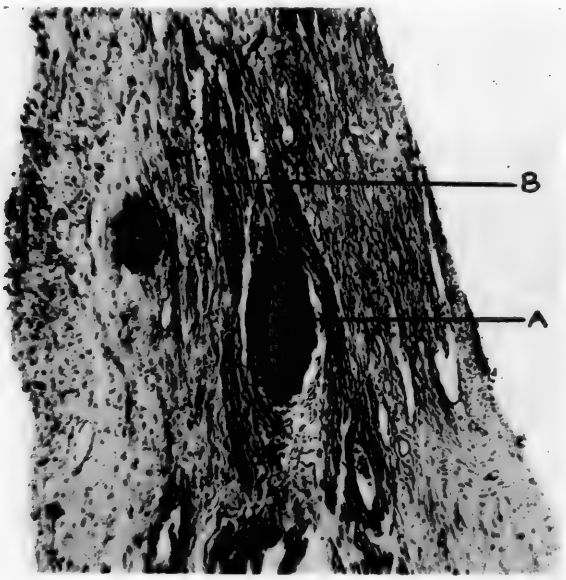


FIG. 7. DENTAL PULP: CALCIFIED AREA (*A*) WITHIN NERVE TRUNK (*B*)
This tooth was non-carious, but deeply involved in pyorrhea. It shows calcification engaging nerve trunks, and possibility of reflex neuralgia arising from pyorrhetic teeth.

and extensively in pyorrhetic teeth where caries never existed (*fig. 6*). In itself calcific change may never do any harm, but many of these calcifications impinge upon and engulf nerve fibres in their formation and thus cause reflex neuralgia (*fig. 7*). Besides calcification the nerves of the pulp are almost always involved by inflammatory changes that take place in the surrounding tissue. These nerves are so abundant that any change within the pulp must affect them. For this reason

the tooth pulp is the greatest source of obscure facial pain (*figs. 8, 9, 10*).

Another dental cause of facial neuralgia, and one to which perhaps too much importance is given, is the totally embedded tooth. It is

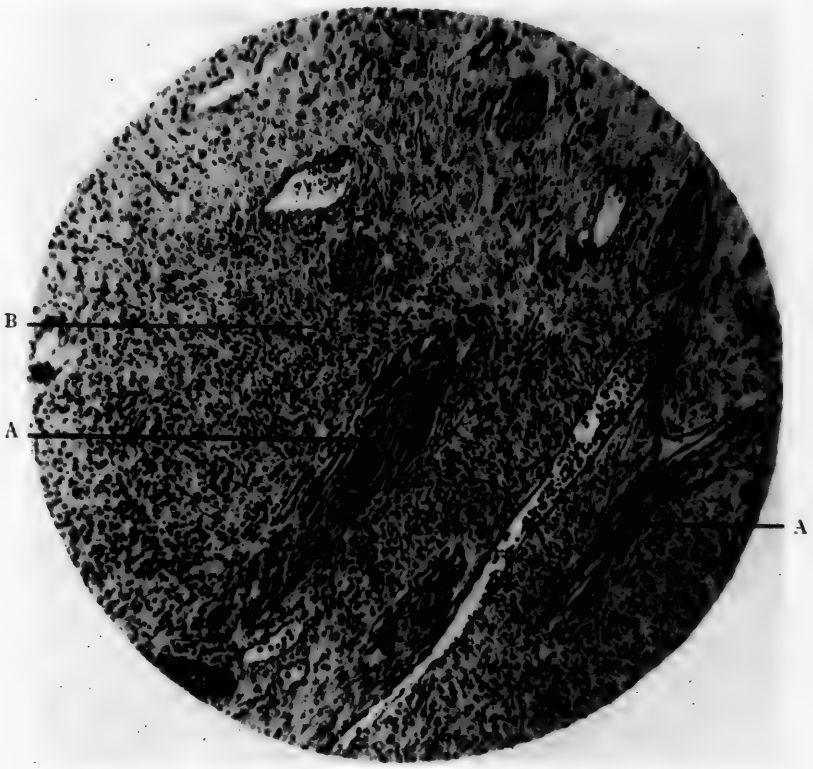


FIG. 8. PULPITIS AND INTERSTITIAL NEURITIS OF PULPAL NERVES
*A, A*, nerve trunks in inflamed area. *B*, zone of inflammation

a popular belief that these teeth press on nerves and thus cause all kinds of serious conditions. This theory is untenable and there is no true basis for its acceptance. Personally I believe that in the vast majority of cases embedded teeth cause no pain, but do occasionally produce other nervous disturbances. Possibly in the instance of the

upper, embedded, cuspid tooth the calcifications so often found in the pulp may cause reflex pain, because of the impingement of these calcified areas on the nerves of the pulp, but in this instance the pain is due to a condition within the tooth and not to the abnormal position. The partially erupted and impacted tooth causes mischief, but probably only indirectly. The lower third molar is most often impacted. Frequently, with the overlying gum, it forms a cul-de-sac in which infection takes place. Pain is usually radiated to the ear

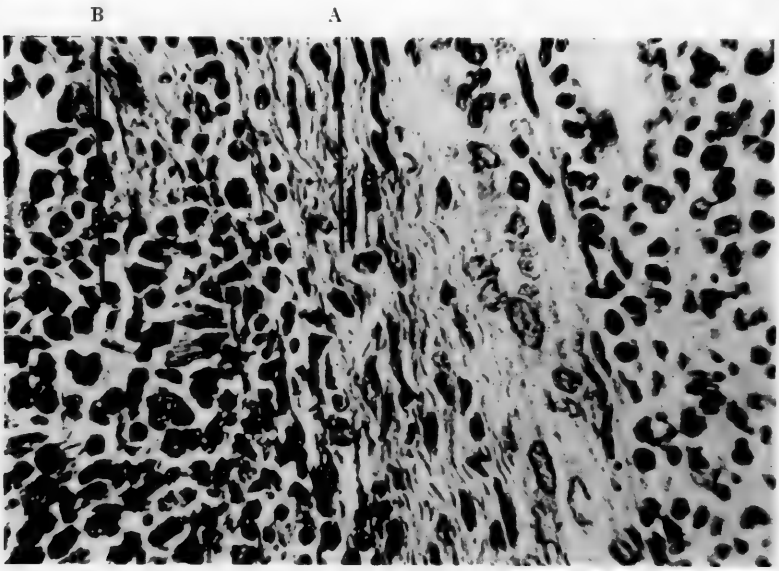


FIG. 9. ACUTE PULPITIS AND INTERSTITIAL NEURITIS
*A*, nerve trunk infiltrated with inflammatory products. *B*, area of inflammation

from such an infected gum flap. At other times the pressure of an erupting wisdom tooth coming through either normally or abnormally gives rise to intense reflex neuralgia. Again, a tilted lower third molar, in attempting to erupt, presses against the adjacent second molar, causing pressure dissolution of the hard structure of this tooth and pulp exposure, with its consequent train of reflex symptoms.

In the upper jaw the bicuspids are occasionally crowded out of the arch and are placed medially. The gum over the roots of these teeth

is stretched and compressed. Normally the gum of the palate has a pad of fat beneath the corium. In this fat are a large number of nerves. In removing one of these palatally-placed bicuspids from a patient with an unaccountable earache, I also cut away the surround-

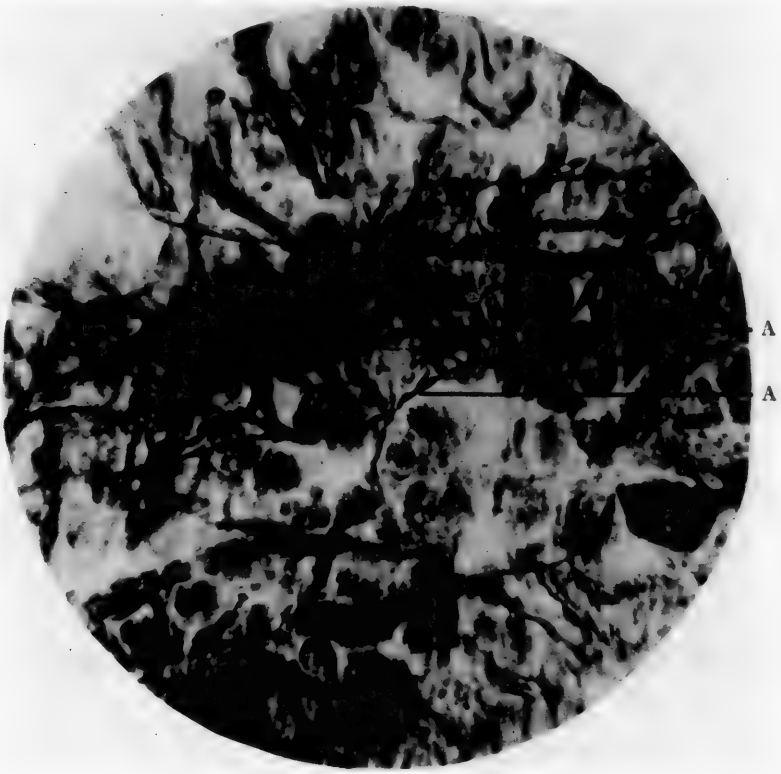


FIG. 10. DENTAL PULP

Specimen stained by writer's modification of Cajal-De Castro technique. *A*, *A*, nerve fibers in gangrenous field. See *fig. 12*.

ing gum. A section of this tissue disclosed the total disappearance of the fat pad, and a marked distortion of the few nerves in the corium.

One more cause of facial pain lies in infection and traumatism of the alveolar process. The text books speak very little, if at all, about

the innervation of this structure (*figs. 11* and *12*). In fact, its neuroanatomy has been so grossly neglected that probably no one considers its nerve involvement in operative procedures and paradental infections. Operations, such as the common one of tooth removal, may traumatize the nerves of the alveolar process to such an extent that chronic reflex neuralgia results. Also nerves may be affected by scar formation, with the same sequelae. The severe reflex-pain accompanying an infected tooth-socket is the result of interstitial neuritis of the nerves of the alveolar process. Obscure facial pain is sometimes

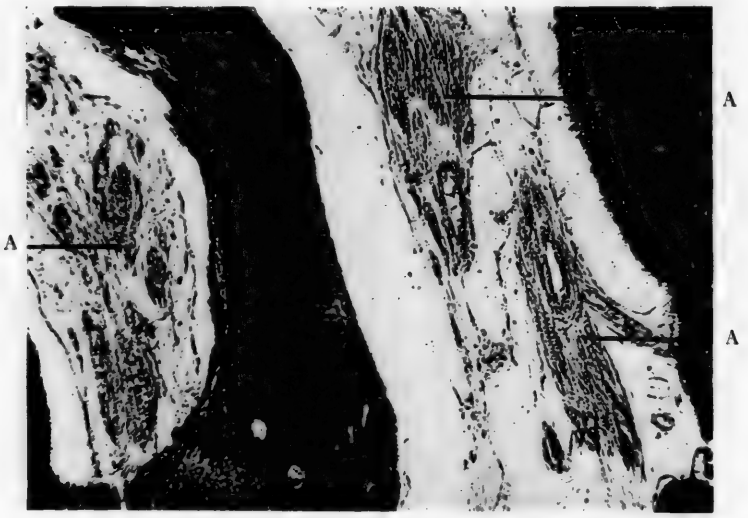


FIG. 11. NERVE TRUNKS (*A*, *A*, *A*) IN MARROW SPACES OF ALVEOLAR PROCESS

found associated with pyorrhetic conditions, and is frequently the result of infection of the process nerves. The possibility of injury to the large nerves of the jaws needs attention. Dr. Dorrance reported a case of neuroma of the lingual nerve following the removal of a lower molar. I know of a case of neuroma of the mental nerve resulting from the extirpation of a small cyst in the lower bicuspid area.

Chronic pain in the tongue is frequently due to traumatic injury of the lingual nerve, or its filaments, during the removal of a lower

molar or bicuspid. It may result also from a mandibular injection in which the lingual nerve is injured during the anesthetic procedure.

FIG. 12. HIGHER ENLARGEMENT OF *fig. 10*
*A*, neurokeratin of nerve fibers (*A*)

# CONCERNING THE PERMEABILITY OF HUMAN ENAMEL[1]

EDMUND APPLEBAUM, D.D.S.

*Laboratory of Histology and Embryology, School of Dental and Oral Surgery, Columbia University, New York City*

For many years considerable discussion has arisen as a result of the work of various investigators concerning the question of enamel permeability. The opinions of these men have been reviewed in recent publications. A thorough and cautious consideration of the evidence presented seems to warrant a belief in the permeability of the enamel. However, this permeability is not a very simple matter, since it involves the question of membrane charge and pore-wall phenomena. Any discussion of the permeability of the enamel must, as a result, take cognizance of its complexity as a membrane structure. It is probably because of the electrical properties of the enamel that many workers have been disappointed in their attempts to force dyes into the structure. Then again, the failure in these experiments was due, possibly, to the fact that the color ion of the organic dye was too large to penetrate the small channels in the enamel.

In the experiments described in this paper, the writer tried to delineate by a staining method the channels in enamel through which a salt or water might pass during the process of electroendosmosis. This is simply a movement of water through the pores of a membrane with the application of an electric current. Many considerations are involved in this conception of the enamel membrane. Bechold[2] introduces the conception of a membrane by discussing diffusion

"If pure water is layered over a concentrated sugar or salt solution so that there is no mixing it is found that after a time the sugar or salt diffuses into the water. It is, in principle, the same process as when one permits a

[1] Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund of New York in the pursuit of this study.

[2] Bechold: Colloids in Biology and Medicine, p. 52, 1919; D. Van Nostrand Co., New York City.

compressed gas to stream into the air—the distinction lies only in the difference in speed.

"In the organism diffusion occurs in a more or less dense colloidal medium. When the concentration of the colloid is not very great the diffusion is not much impeded. If we make the colloid medium thicker and thicker, i.e. poorer in water, diffusion must be increasingly hindered. We very soon reach a point where no colloids are able to diffuse into it and we have a special case, the membrane.

"Since the time of Traube, artificial or precipitation membranes have been studied. The chief interest centered in the osmotic phenomena of salt solutions which could be investigated with the aid of such membranes. They are briefly described as semi-permeable membranes because they are permeable to water though impermeable to most crystalloids.

"There are membranes of very different permeabilities. Some are permeable for a salt solution on either side, others prevent the diffusion of both salts. The influence of electrolytes on the membranes of the organisms and their permeability may depend on different causes. Differences of potential may develop at the interface between a solid phase and a fluid. Thus, for example, cellulose and wool are negatively charged with respect to pure water. The presence of salts likewise raises or lowers the difference in potential."

A membrane may thus be the seat of an electro-motive force when it separates two salt solutions of different concentrations. Klein and Amberson have presented electroendosmotic evidence that human enamel is a permeable membrane. Similar phenomena were observed by Bunting, Livingston, and Entin. Klein and Amberson[8] wrote:

"Water in the pores of an organized membrane behaves as if it had assumed an electrical charge opposite in sign to that resident upon the pore walls themselves, and that with an externally applied potential difference water may be made to pass through the structure of the membrane. The movement of water thus produced is spoken of as electroendosmosis.

"Several unsuccessful attempts were made to secure an electroendosmotic transport of water across enamel cups before we finally were able to secure the crude measurements here reported. The amount of water transported even with high voltages is, however, very small. We have been able to obtain only very rough qualitative indications of the direction of water

[8] Klein and Amberson: A physico-chemical study of the structure of dental enamel. *Journal of Dental Research*, 1929, ix, p. 667.

movement under various experimental conditions, but these are sufficient to justify certain general conclusions."

In the summary of the paper, Klein and Amberson stated:

"The slow movement of water across enamel as a result of an osmotic gradient is also demonstrated under conditions free from technical defects in previous work.

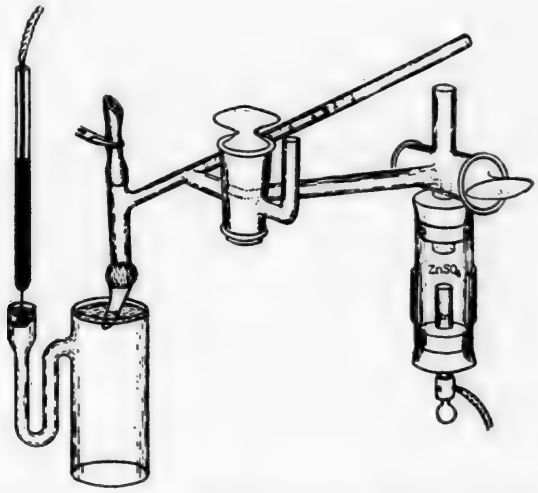


FIG. 1. Apparatus for transporting $HgCl_2$ through enamel cup by electroendosmosis (Klein and Amberson, *Journal of Dental Research*, 1929, ix, p. 677; Oct.)

"These observations do not permit the identification of the pathways through the enamel structure by which water and salts penetrate. It is suggested that the movements probably occur along the organic matrix between the enamel rods described by Malleson and by Bödecker."

The writer has attempted to repeat part of the work of Klein and Amberson and to determine "the pathways through the enamel structures by which water and salts penetrate." Both of these investigators have greatly aided in the work. Their help is gratefully acknowledged. The apparatus used for the electroendosmosis was similar to that employed for the same purpose by Klein and Amberson,

but the KCl solution in the enamel cup was colored with an anilin dye. The dye was intended to stain the enamel during the outward movement of the solution through it under the influence of the applied current of 200 volts, and also, in the presence of cracks in the enamel cup mentioned below, *by grossly flowing through the enamel*, to reveal

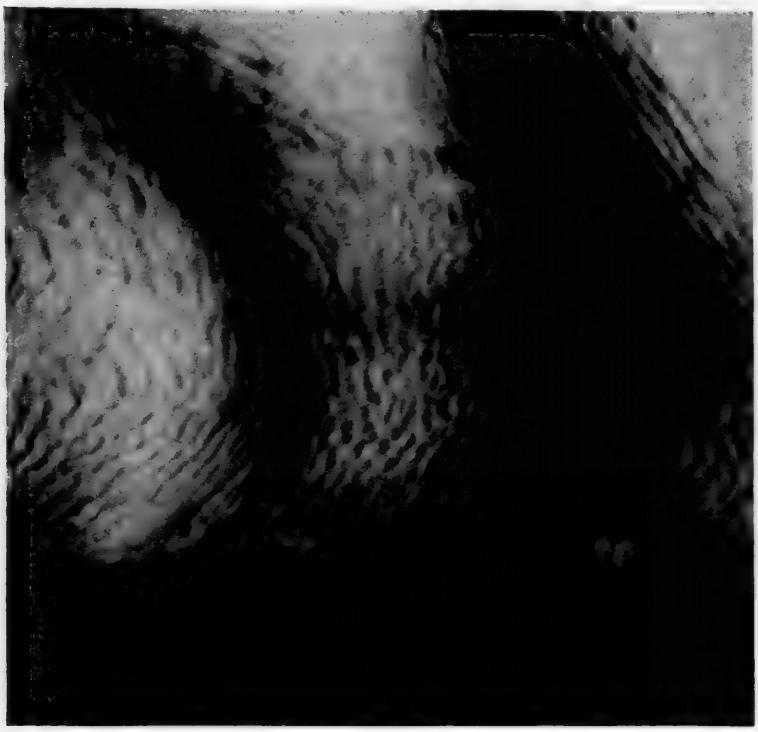

FIG. 2. Photomicrograph of "organic matrix" of enamel in relation to dentin prepared by celloidin decalcifying method of Bödecker. Stained with haematoxylin-eosin. Enlargement: 450×

these defects at once. Methylene blue, safranin, and fuchsin, the anilin dyes thus used, served the latter purpose, but none of them diffused into the enamel.

Undoubtedly many factors are involved in the staining of enamel by the method of electroendosmosis, among them being (a) the tooth

itself, especially the age factor—that is, whether erupted or non-erupted; (b) concentration of the solutions; (c) size of the ions of the dye; and (d) length of time the current is applied. The accompanying photomicrographs show a human bicuspid, *unerupted* but completely formed, that was stained by mercuric bichloride used in the electroendosmosis. Mercuric bichloride was chosen, after the

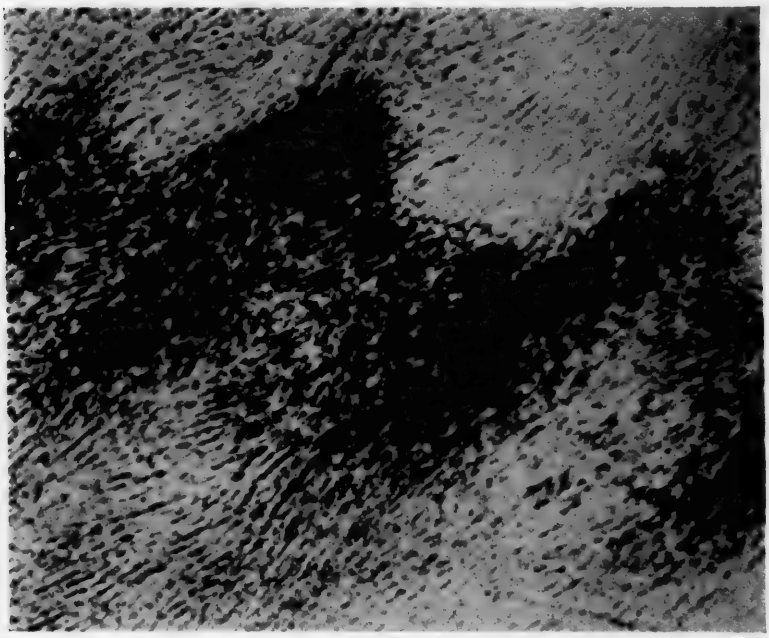

FIG. 3. Photomicrograph of ground section of enamel cup stained by electroendosmosis. Stain (produced by mercury) is confined to interprismatic substance—"organic matrix." Enlargement: 200 ×

failures with the dyes, because it was believed that a representative metallic salt could be driven through the enamel "pathways" more easily than the large ions of an anilin dye. This opinion seems to have been confirmed by the result. An *unerupted* tooth was used because teeth become denser after eruption.

The root of the tooth was cut off with a carborundum disk. The crown was excavated with various sized round burs to remove most

of the dentin. The enamel cup was sealed with dental "sticky" wax to the end of the glass tube in fig. 1.[4] The solution of $HgCl_2$ was then

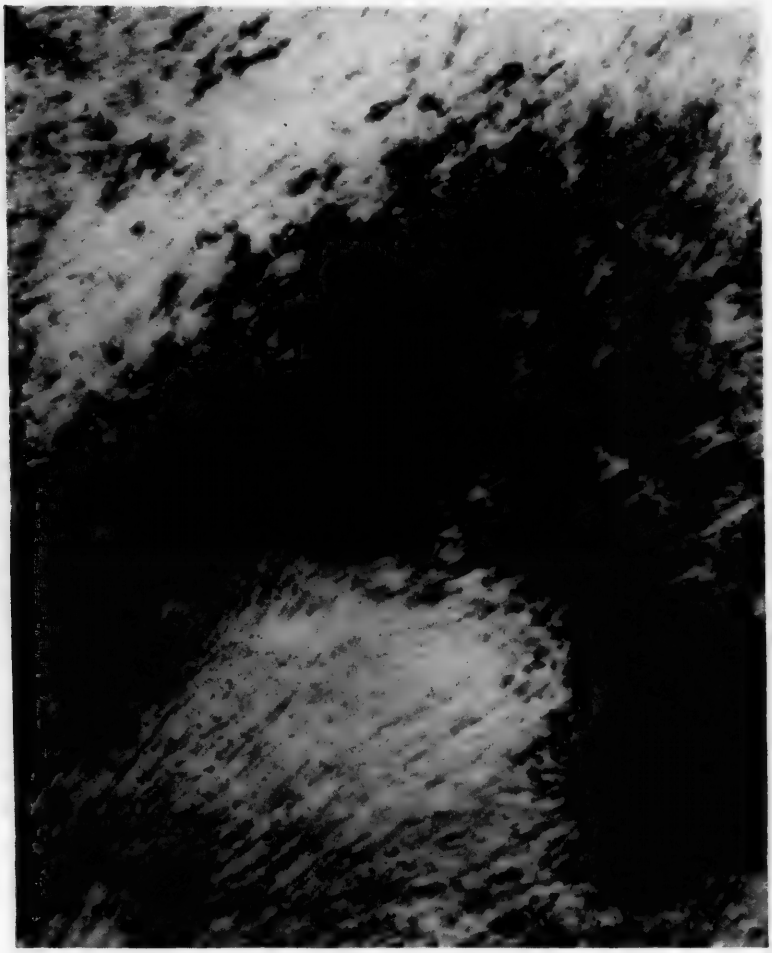

FIG. 4. Higher enlargement of fig. 3. Enlargement: 450 ×

poured into this tube, which was connected with a Zn-$ZnSO_4$ electrode and the enamel cup. The tip of the tooth was placed in contact

[4] Klein and Amberson: *Loc. cit.*

with a saturated solution of KCl in the lower vessel, and electrical connections were completed through a platinum electrode dipping into the side-arm of the vessel. Then a current of 200 volts was

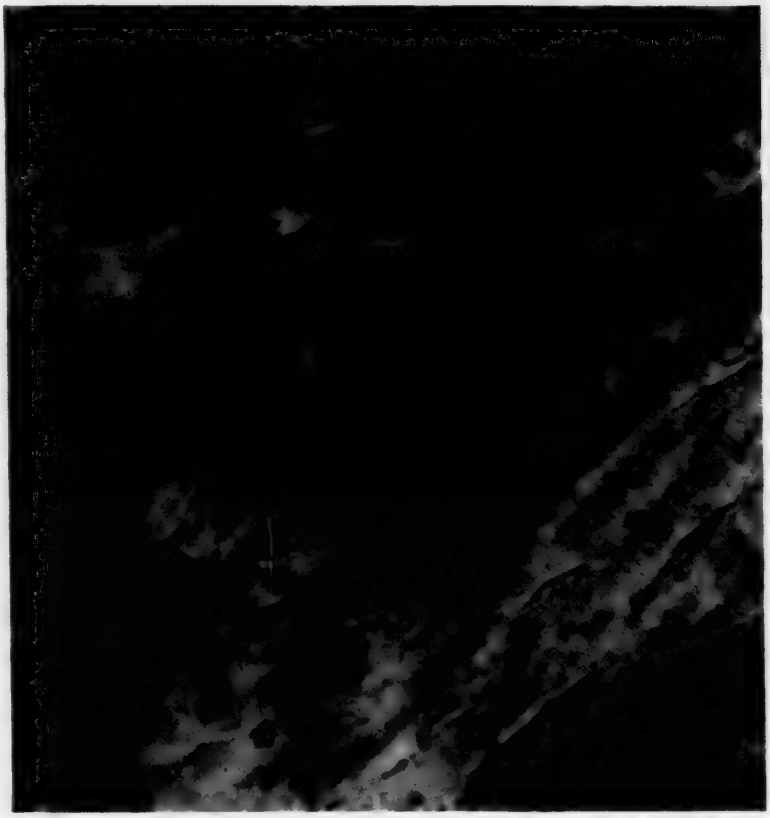

FIG. 5. Oil-immersion photomicrograph of stained enamel. Enlargement: 2000 ×

applied to the two terminals, the inner surface of the enamel cup having been connected to the anode and the outer to the cathode.

The tooth shown in the photomicrographs was kept on the apparatus two weeks to secure the staining of the "pathways." *Fig. 2*, a decalcified cross section of a human tooth, shows the relation of the organic matrix of the enamel to the dentin. *Fig. 3* (ground section,

200 ×) shows the peculiar black wavy stain on the buccal plate of the enamel cup. *Fig. 4* is a higher enlargement (450 ×). *Fig. 5* (oil immersion, 2000 ×) indicates the appearance of the stained enamel. The enamel rod when seen in sharp focus is not stained; only the interprismatic substance—the organic matrix—is stained. The manner in which the Hg ion stained the enamel has not yet been determined.

An interesting sidelight is thrown on the phenomena of electroendosmosis by a chemical analysis of the salts in the lower vessel—outside the enamel cup. Zinc was present in the solution. Since there was no leak—as shown by the failure of the associated dye to flow to the exterior liquid—zinc must have come through the enamel from the solution of $ZnSO_4$ in the Zn electrode.

It is outside the field of the writer's experience to explain the complex physical and chemical problems entailed here, but the experiments seem to substantiate the findings that the enamel is permeable; and also to indicate that the "pathways" for the passage of a fluid can be stained when a suitable dye is used as well as a suitable tooth.

# RESISTANCE TO CARIES[1]

THEODORE B. BEUST, D.D.S., M.D., F.A.C.D.

*School of Dentistry, University of Louisville, Louisville, Ky.*

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## I. INTRODUCTION

Resistance, when expressing a quality of a tissue, may depend on a variety of factors. Resistance to microörganismal or other cytoplasmic irritations consists of a complex of cellular phenomena attended by modifications of the quality of the blood; or, it may be due to a heightened aggressiveness of the organized defence-units of the body, which again are dependent on certain qualities of the blood. Resistance exhibited by the skin or mucous membrane to some of the halogens, or to arsenic, demands other interpretations, whereas the resistance offered by the tooth to caries, assuming the chemico-parasitic theory to be correct, would involve a physical resistance to, or a neutralization of, acids by the tooth.

Resistance, it must be borne in mind, is not absolute, but relative. It is always possible that an increase in the potency or the attacking factors may break down any resistance. Many orders of mammals, including the Primates, are known to be caries-susceptible. Individual members of these orders, proving themselves through some

[1] Work supported, in part, by a grant from the Research Commission of the American Dental Association. Read at the Ninth General Meeting of the International Association for Dental Research, Memphis, Tenn., March 21–22, 1931; *Journal of Dental Research*, 1931, xi, p. 469; June.

unknown cause to be caries-resistant, exhibit therefore what is known as the acquired form of resistance. In calling this form acquired, we must disregard for the time both the naturally acquired and the artificially acquired forms known to immunology, for we have as yet no reason to believe that an attack of caries confers future immunity, nor have we learned to immunize against caries. We are justified it appears, in the interest of clarity, in framing a new division of these varieties especially applicable to caries. One form, as above intimated, may be regarded as an acquired *serologic* type, a resistance not depending on condition of tooth substance; another, depending on the latent or potential powers of resistance in certain molecular combinations or physical constitution of tooth substance, could appropriately be termed *physical* resistance. This last form is, of course, also an acquired resistance. The justification for such a division will be developed as we proceed with this study.

The dental profession has come to recognize certain periods of caries susceptivity, e.g., a period extending from early childhood to a time near the age of thirty. This is normally followed in the human subject by a condition of relative immunity, extending well into the age of senescence. During the immune period exacerbations to the state of susceptivity may occur, as is seen during pregnancy or during other periods of stress. The object of this paper is to discuss one of the factors that apparently contributes to the state of relative resistance now generally recognized. It is furthermore intended to show the limitations of certain hypotheses advanced to explain the cause of caries immunity.

## II. PERMEABILITY AND SCLEROSIS OF THE DENTIN

Movement of fluids through the unobstructed dentinal tubules is too well known to be considered here. Dentin ground-substance, as I have pointed out is, in the early stages of development, colorable by dyes. This colorability is gradually lost, first, through increased sclerosis of the ground substance; later, by gradual closure of the tubules through deposition in the lumina. This closure is attended by loss of colorability of the tubular contents (7, 12, 13, 14). As successive additions of dentin are laid down, progressive contraction of the pulp cavity occurs. This contraction often leads to complete ob-

literation of the pulp. There are many indications that the fibrils, which are automatically dissociated in this process, are physiologically displaced by stain-resisting matter. Complete loss of all of the colorable soft tissue of the tooth may result. We then have the completely matured tooth, in fact a solid mass of tooth substance. The age at which such a condition should normally be attained is difficult to conjecture inasmuch as our concept of the normal span of life, ranging from three-score years and ten to the patriarchal estimate of nearly a millenium, permits much latitude for speculation. However this may be, the amount of sclerosed dentin in sound teeth, as attested by staining experiments, is correlated with the degree of contraction exhibited by the pulp cavity. Specimens exhibiting practically complete sclerosis of the entire anatomical crown are not at all uncommon. *Figs. 1, 2* and *3* give an idea of the distribution of the sclerosed tissue.

That the stain-refusing or chromophobic tissue is really sclerosed is proved by the following experiment. The tooth shown in *fig. 4*, after having been halved, was stained for one month. For the staining method see the bibliography (13). The result is indicated in the photograph. The color in the neighborhood of the pulp cavity evidences the presence of some open tubules. The worn pit on the morsal surface was removed by grinding, whereupon the crown was sectioned transversely, as indicated by the cross lines, into apical, median, and basal sections. It is obvious that this operation opened the tubules. The median section, *fig. 5, a*, was ground and mounted without further treatment; the apical section, *fig. 5, b*, was exposed to alcoholic fuchsin-stain for one week, and the basal section, *fig. 5, c*, was exposed to alcoholic Bismark-brown for a similar period. Any uncalcified organic matter contained in the tubules opened by the section would have exhibited coloration throughout. This did not occur, as may be seen by referring to the photograph. The slight degree of susceptivity to stains in sections *b* and *c* could already be seen in the original photograph.

I should like to call attention to some new evidence concerning the formation of sclerosed areas under slowly advancing or arrested caries. The transparent zones need no further rehearsal here (12). It will be seen from *fig. 6* that the transparent zone is not the whole story. At the pulpal side of the transparent zones (*fig. 6, b, b, b*),

we almost regularly find tissue modifications that are identical morphologically and in reaction with the zones seen under erosions and

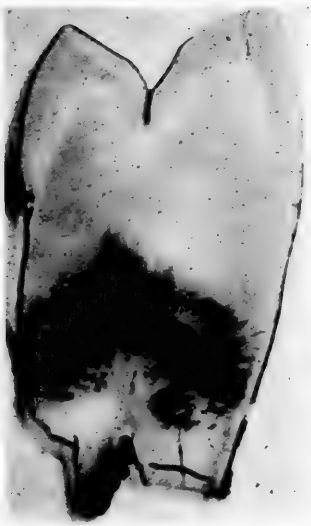

FIG. 1 FIG. 2

FIG. 1. GROUND SECTION. REFLECTED LIGHT

After removal of root-apex, tooth was stained in alcoholic fuchsin for one month Sections were immersed in stain for one week. Penetration by stain is limited to middle one-third of tooth. Crown completely sclerosed.

FIG. 2. HALVED TOOTH, STAINED AFTER GRINDING. REFLECTED LIGHT

Exposure to stain, ten days. Crown and part of root are sclerosed. Shaded tissue seen at amelo-dentinal junction in *figs. 2, 3,* and *4,* is transparent dentin.

in the dentin of aging teeth. They are the expression of reactions on the part of the tooth to irritation. Such zones, as I have pointed out

(12, 13, 14), appear dark by ordinary microscopic examination and have often been regarded as extensions of the caries process toward the pulp, whereas in reality they consist of intact, physiologically

FIG. 3 FIG. 4

FIG. 3. HALVED TOOTH. REFLECTED LIGHT

Stained after grinding. Exposure to stain, eight days. Nearly whole of crown consists of sclerosed tissue. See *fig. 2*.

FIG. 4. HALVED TOOTH. REFLECTED LIGHT

Stained after grinding. Exposure to stain, one month. Cross-sections were prepared as indicated by transverse lines. Compare with *fig. 5*. See *fig. 2*.

modified tissue. Photographed by reflected light, *fig. 7*, their true nature is revealed. They correspond to the zones usually found under perfect fillings in healthy teeth. I discussed the significance of

FIG. 5. SECTIONS PREPARED FROM TOOTH SHOWN IN FIG. 4. REFLECTED LIGHT

*a*, medial section. *b*, apical section. *c*, basal section. Consult text for further description.

FIG. 6. STAINED CARIOUS MOLAR. TRANSMITTED LIGHT

+, carious tissue. *a*, *a*, transparent zones. *b*, *b*, *b*, sclerosed opaque dentin, here appearing dark. Compare with *fig.* 7.

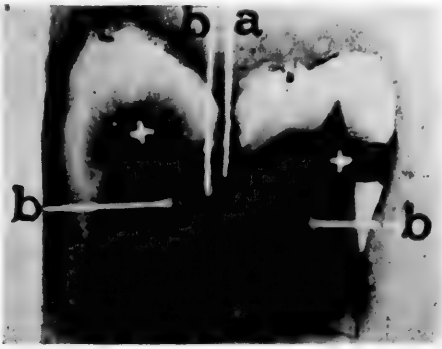


FIG. 7. SECTION SHOWN IN *fig. 6*. REFLECTED LIGHT

Lettering is same as for *fig. 6*. Sclerosed opaque tissue here appears white.

these zones in 1918 (7). It would not embarrass me should some prefer to regard these areas as pathologic. There can be no doubt that such zones are areas of resistance expressing a high degree of vitality resident in the tooth, that they consist of sclerosed tissue, and that they are chromophobic.

### *Discussion of stains*

The stains were selected rather empirically. Following the example of Caush (18) I used basic fuchsin in my earlier experiments and have continued its use since. This stain appears to be the most practical for ground sections, as its color deviates markedly from those of the natural stains. Moreover it is quite permanent. Fear of the imputed acid radical is ungrounded. Any decalcifying action would necessarily be counteracted long before the stain had wandered the length of the tubule. Unstained dentin in immediate contact with the stain may be found in the sections. In fact sclerosed tissue may be immersed in the solution for weeks without showing decalcification or discoloration. The fact that the stain exhibits selectivity, even after extended exposure, disproves the presence of free acids. Assuming that this stain does decalcify then, I may ask, why do the older tissues refuse it? But there is yet another point to be considered in the staining of dentin and enamel. We are not, in these tissues, dealing altogether with affinities. We are dealing with sieve-like structures permeated by capillary spaces. Whether or not these spaces contain protein substances is of no consequence, as experience with many hundreds of teeth has shown that in the young state they permit diffusion of stains into their lumina. Solutions on equimolecular footing with the contents of the spaces will not readily diffuse into them. To increase the desired interchange we must alter the pressure. That is one of the advantages in the use of alcohol as an ingredient of the staining solutions. It is a fixative that will not decompose the inorganic parts of the tooth, reduces surface tension, is a solvent for the dye, and dissolves some of the fats. Conditions for active penetration are favorable when water-soaked teeth are suspended in the upper layers of the solutions. I would again insist that unaltered or empty tubules, or tubules containing necrosed fibrillar substance, will positively stain when subjected to the methods here practised.

## III. ANATOMY OF THE ENAMEL

The generally accepted erroneous views concerning the anatomy of the enamel have been a bar to progress in caries investigation. I

have repeatedly pointed out (8, 9, 10), that the descriptions in our text-books on dental histology are grossly misleading. This misconception of structure is reflected in current biologic misconceptions. Speaking more specifically, the figures called tufts, now almost universally regarded as defects, were shown by me to be a part of a great system of tubes, anatomically arranged and traversing the enamel

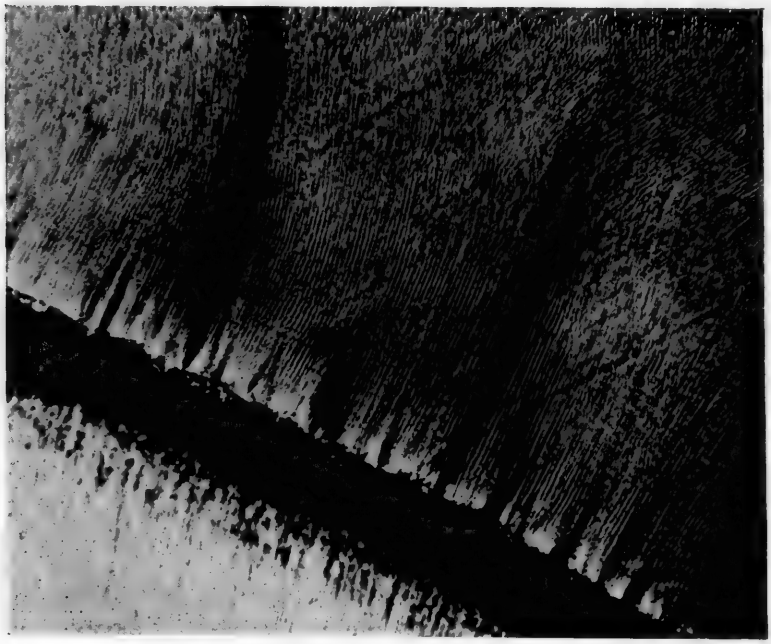

FIG. 8. STAINED INCISOR OF COW. TRANSMITTED LIGHT

Stained by capillary attraction. Alcoholic basic-fuchsin used. All tufts and much of inter-rodal substance show penetration. Halo of red color surrounds each enamel figure. In order to outline dentinal tubules, dentin was shaded in making print.

from the tip of the crown to the region near the amelo-cemental junction. This system has direct bearing on our problem. Orban's account of the tufts (21) partly corroborates my views, while Bödecker's description of the morphology of the tufts (17) is erroneous. Lines of tufts divide the inner regions of the enamel into parallel compartments (8, 9, 10). Cross sections of the enamel rods, which

include these regions, invariably display such a division. No one can produce a tooth anterior to the molars that does not exhibit this arrangement. A cross section of a tooth of average size that fails to show a hundred or more tufts, characteristically arranged, does not exist. Proof can never be submitted that these tufts are not cross-sections of the parallel rows described.

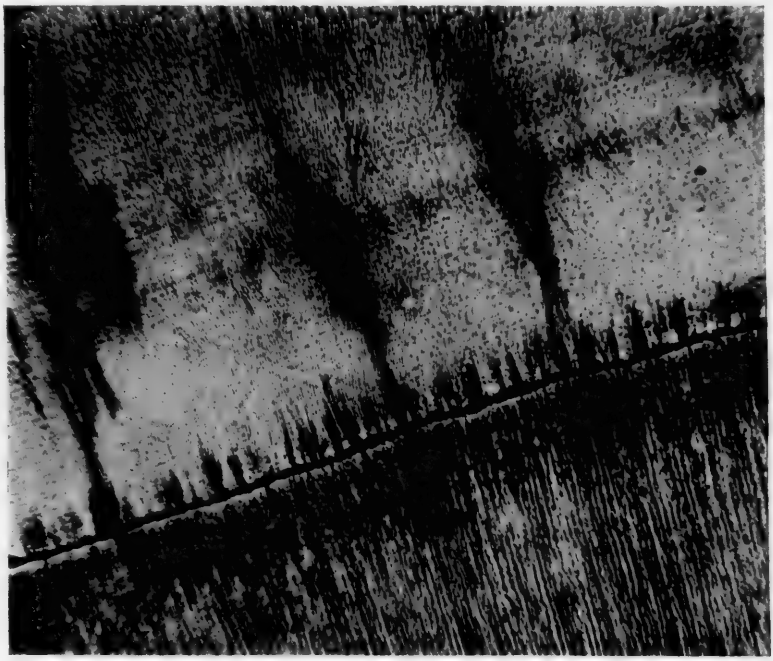

FIG. 9. MOLAR OF PIG. TRANSMITTED LIGHT

Stained by capillary attraction with alcoholic basic-fuchsin. Tufts and inter-rodal substance display penetration, as in *fig. 8*. Dentin was shaded in making print.

## IV. PERMEABILITY AND ULTIMATE SCLEROSIS OF THE ENAMEL

Young enamel-rod-substance, up to a time shortly before normal tooth eruption, is colorable by stains (11). I recognized and published the fact, in my early work on this subject (11, 5, 6, 9), that the more centrally situated prism substance in newly erupted teeth has affinity for dyes. With increasing age this rod-colorability disappears,

indicating further enamel sclerosis after eruption. Following this stage the inter-rodal substance continues to conduct stain for a time. The morphology of the cross-sections of the stain-conducting canals was described by me in 1912 (4, fig. 3) as follows: "The canals transmitting the bulk of the stain are seen to consist of peculiarly shaped fissures lying between the prisms. The finest canals present themselves as half-moon-shaped lines partially enveloping the enamel rods." This was the first attempt ever made to characterize the tract travelled by the diffusional currents. Flow through the last-mentioned "finest canals" ordinarily becomes undemonstrable, soon after eruption in the human subject; but, during a long period of early life, penetration of the tuft systems can be demonstrated in the teeth of man

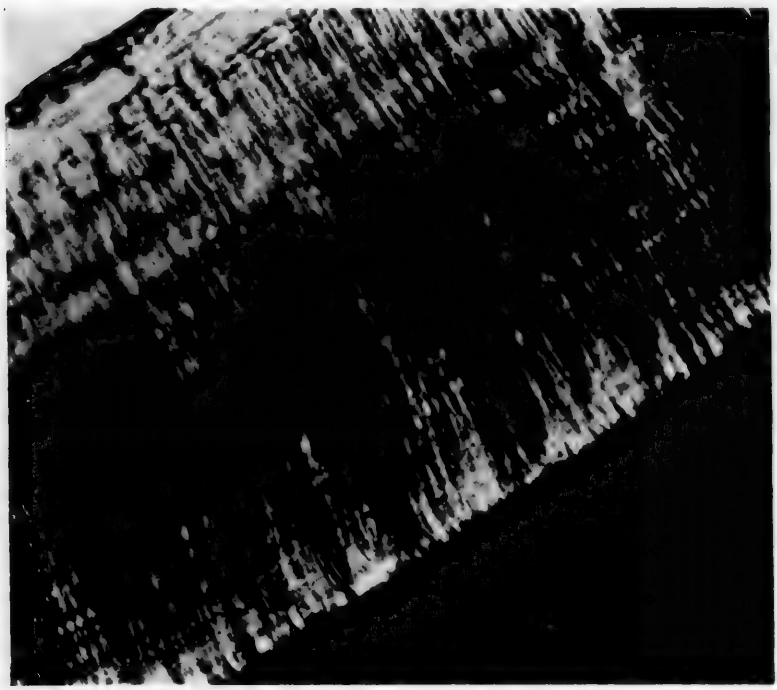

FIG. 10. MOLAR OF SKUNK. TRANSMITTED LIGHT

Stained by capillary attraction. Shadows in enamel correspond to penetration of dye.

(4, fig. 1). In the lower animals demonstration of the flow is also possible. *Fig. 8* shows a specimen of bovine enamel. *Fig. 9* shows enamel of a pig. *Fig. 10* shows enamel of a skunk. A study of the legends under these illustrations must convince one of the occurrence of fluid interchange between the pulp and the enamel. In all of these cases the teeth were stained first, and afterwards sectioned. The stained figures in the enamel are therefore not artifacts. The permeability of these figures is gradually lost with increasing age. It extends well past the period of adolescence. Finally a complete sclerosis, barring a few open tufts and lamellae here and there, is found to occur. This precedes the sclerosis of the dentin. There can be no doubt that the enamel is a conductor of impulses, or of irritation if one chooses to use that term. Dentin sclerosis occurs, in anticipation as it were, before caries or progressing erosion reaches the amelo-dentinal junction. I have repeatedly seen evidences of that.

Of great significance for our subject is the fact that with increasing age, the great systems of parallel rows become impenetrable, and filled with a substance which is not colorable. Even assuming that these systems are defects, the changes that occur therein positively prove the presence in enamel of phenomena of an anabolic nature. Defects, which experience has amply proved are colorable in early life, should likewise be colorable (excluding anabolism) in adult life. The cessation of the flow of lymph from the pulp may account for the inability of Rosebury and Gies (22) to detect the presence of diffusible protein in the enamel.

## V. ENAMEL METABOLISM

Experimental proof of the possibility of metabolic changes in the enamel has been presented by Caush (18), myself (4), Mummery (19), Bödecker (15), and others. Until very recent times the term metabolism, as applied to the enamel, was meant to signify that tissue changes, induced by physiologic means, occurred in the enamel. Newer conceptions would see the inclusion of catabolism, as well as anabolism, under this heading. Some even display such perversion in their conceptions of the problem as to demand proof of cellular participation and visible regeneration of enamel rods. Constructive

criticism, confined to the publication of experiments substantiating claims, would relieve much confusion in this field. Personally I am convinced (11, p. 2031, fig. 17) that acid-resistant infiltrations from the dentinal tubules diffuse into the inter-rodal substance, the tufts, and the lamellae. The occurrence of such phenomena acquires substantial support from the proof of the progressive infiltration of the dentinal tubules, forming the sclerosed zones, recently presented by me (12).

A thought of an occurrence of catabolic changes in the enamel has never been entertained by me. Current views however lead one to infer that in impairment of the calcium-balance the teeth, together with the bones, are depleted of their inorganic content. As caries susceptivity is supposed to increase in such periods we must infer that a depletion of the enamel is also included. That of course would indicate an enamel catabolism. I have seen no histologic indication of such changes, although I can conceive of such a phenomenon so long as the pulp is large and the dental tubules and tufts are open.

## VI. THEORIES OF RESISTANCE TO CARIES

In the opening chapter of this paper I suggested the division of resistance into an acquired serologic resistance, and into a physical resistance, depending upon the constitution of the tooth. As has been shown the enamel and dentin of youth are permeable and accessible to infiltrations from the pulp. Efferent circulation, or diffusion into these tissues at this age, is certainly as active as any centripetally advancing caries agent. Any resistance exhibited by the tooth under these conditions would inferentially be of the acquired serologic type. About twenty years ago I dared to write a number of contributions citing various inconsistencies in the chemico-parasitic theory (1, 2, 5). Some of my objections have since become common property. In 1912, after having excluded the possibility of all flow of stains from without, I proved that stains applied into the pulp cavity could diffuse into the enamel (4). This result justified the view that immunity to caries was explainable by reactions occurring within the tooth. In the summary of this paper I contended that "the enamel is vascular to a varying, sometimes an extreme degree;" also, "the vascularity of the enamel renders it possible, even probable, that immunity to caries

observed in some teeth lies in the tooth itself, and is the result of the formation of anti-bodies." In 1926 Mummery presented a brilliant contribution (20) showing the formation of zones of resistance in the enamel in all but two of twenty cases of incipient caries examined by him. These results were due to the presence of vital forces in the enamel. Very recently Bödecker (16) availed himself of a hypothesis similar to mine. Barring one detail Bödecker's theory is founded on the principle formulated by me in 1912. A difference lies only in the conception of the buffer substance. Where I thought of the potency of anti-bodies of the nature of anti-ferments (3), Bödecker conceives of the neutralization of the acids of decay by the dental lymph.

After the cessation of flow from the pulp through the dental tissues neither of these theories, so far as we can now perceive, explains resistance. Excluding the mixed saliva as a factor, it is then that only the latent or physical form of resistance remains active. Without verifying my assertions for the moment, I feel no hesitancy in stating that a sclerosed tooth-crown is harder to section, harder to grind, and more resistant to various chemicals, than the crown of a tooth not so modified. This phase of the work is not yet ready for publication.

It may be pertinent to outline here, in round numbers, the results of an examination of seventy functionally intact, unselected stained teeth, nine of which contained small fillings:[2]

26, or 38 per cent exhibited sclerosis of over one-half of the anatomical crown.

24, or 35 per cent exhibited sclerosis of one-fourth to one-half of the anatomical crown.

12, or 18 per cent displayed only very slight macroscopic evidences of crown sclerosis.

5, or 7 per cent showed no macroscopic evidences of sclerosis.

(This summary does not take into account the areas composed of transparent sclerosis, as these are not made conspicuous by the method used.)

Approximately 100 per cent of sections of intact adult teeth display some degree of sclerosis.

[2] Infirmary material of unknown history. The teeth were first halved and then stained.

## VII. CONCLUSIONS

In view of the limited amount of work accomplished in this unexplored field, final evaluation of the significance of the observed changes is not yet possible. The observations however appear to justify certain theoretical deductions:

1. A tooth's resistance to caries, or to other tooth-destroying agents, increases in proportion to the amount of its sclerosis.

2. Beyond a certain maximum amount of primary-dentin formation in intact teeth, further contraction of the pulp is ordinarily attended by tubular sclerosis.

3. Sclerosis is an expression of the factors of resistance possessed by a tooth.

4. Hypoplasias, erosions, abrasions, and decay cause a premature formation of sclerosed areas under the seat of the trauma. Secondarily, a hastened contraction of the pulp-cavity occurs in such cases.

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## *Quotations on the Status of Dentistry (continued)*

degree to practise dentistry, the poorest type of technical dental service is to be found. In Switzerland, Sweden, Norway and parts of Germany, where dentistry is taught in conjunction with medical studies, yet independent of medical supervision, as we teach it in this country, the best type of service is found. Based on my own study of the problem in Europe [1926–27], I would both seek and recommend the services of dentists trained in the latter countries during the past decade and would avoid service in the former, unless I knew the dentist and his work personally.—*Guy S. Millberry, D.D.S., F.A.P.N.A., Dean, Dental School, University of California; Pacific Dental Gazette, 1931, xxxix, p. 415; June.*

The assumption that dentistry would be something more or better than it is, if all or a part of it were called "stomatology," exemplifies a readiness to attach more importance to labels than to contents, or more significance to make-believe than to reality. The term "medicine" originally meant to remedy or cure. ["*Medicina* (ars)"—healing art; "*medicina* (res)"—a remedy; "*medicus*"—of healing; also a physician.] Today "medicine," by including measures to *prevent* sickness and by throwing *etymology* overboard, means not only to remedy or cure, but also *quite the contrary*—to make remedial or curative treatment unnecessary. As medical knowledge has advanced and medical procedures have been changed in consonance, including the development of prevention and of surgery, new meanings have been imparted to the term "medicine." But instead of replacing "medicine" by a succession of words intended to indicate *etymologically* the broader or deeper modern meanings, the import of the original term has been conveniently enlarged as the scope of medical practice has expanded. Thus, the term "medicine" has come to mean more and more as *physicians*, without restricting themselves to its *etymology*, have included *more and more in its significance.* Along the same line of reasoning one sees clearly that, although *etymologically* the term "dentistry" refers only to the teeth, nevertheless logically it also comprehends the contiguous parts and their systemic relationships. Certainly by *usage* in conformity with this logic, and also with the criteria of common sense, the term "dentistry" has *acquired* the broader meaning suggested by the more detailed and lengthy term "oral health-service." By logic, usage, and convenience, the term "dentistry" has thus become the *practical* equivalent of "stomatology," which *etymologically* means the sum of scientific knowledge concerning the mouth, but *etymologically does not include the rest of the body* (although "stomatology" is now *used* by some to include systemic relationships with the mouth). This being true, we are unable to agree that "dentistry" and "dentists," which are terms that have long represented the earnest and aspiring endeavors of generations of faithful practitioners of oral health-service, should be formally replaced for *etymological* reasons by "stomatology" and "stomatologists."—*American College of Dentists*, March, 1929.

### Important Books Received

**The teeth and health.** By J. Sim Wallace, D.Sc., M.D., L.D.S., Lecturer on Preventive Dentistry, King's College Hospital; *formerly* Dental Surgeon and Lecturer on Dental Surgery and Pathology, London Hospital. 1926: pp. 214; 5¼ x 3 in.; 8 illustrations; 3s. 6d. net. Faber and Gwyer, London, England.

**Variations in the form of the jaws, with special reference to their etiology and their relation to the occlusion of the dental arches.** By J. Sim Wallace, M.D., D.Sc., L.D.S., Lecturer on Preventive Dentistry, King's College Hospital; *late* Dental Surgeon and Lecturer on Dental Surgery and Pathology, London Hospital. *The Cartwright Prize Essay, 1920–1925.* 1927: pp. 265; 6¾ x 4 in.; 84 illustrations; 17s. 6d. net. Baillière, Tindall and Cox, London, England.

**Studies on the mineral metabolism during pregnancy and lactation and its bearing on the disposition to rickets and dental caries.** By Kirsten Utheim Toverud, M.D., and Guttorm Toverud, Ph.D., Department of Physiology, Dental School of Norway, Oslo, Norway. 1931: pp. 137; 6¾ x 4 in.; 34 illustrations. Acta Paediatrica, Stockholm, Sweden.

**Four lectures on "the aetiology of irregularity and malocclusion of the teeth."** By J. C. Brash, M.C., M.A., M.D., University of Birmingham. With an *Appendix* "concerning orthodontic problems and their solution." By Miss M. L. Tildesley, Research Assistant, Royal College of Surgeons Museum, London. 1931: pp. 274; 7¾ x 4¾ in.; 195 illustrations; 5s. net. Dental Board of the United Kingdom, London, England.

**The cause of mottled enamel, a defect of human teeth.** By Margaret Cammack Smith and Edith M. Lantz, Department of Nutrition, and H. V. Smith, Department of Agricultural Chemistry, College of Agriculture, Agricultural Experiment Station, University of Arizona. Technical Bulletin No. 32: June 10, 1931. Pp. 32; 7¼ x 4¼ in.; 7 illustrations. University of Arizona, Tucson, Ariz.

**The teeth, the bony palate, and the mandible in Bantu races of South Africa.** By J. C. Middleton Shaw, M.A., B. Dent. Sc. (Dub.), D.D.S. (Johannesburg), F.I.C.D., Professor of Dental Science; Dean of the Faculty of Dentistry; Director of the Dental Hospital; and Lecturer in Dental Anatomy, Physiology and Histology, University of the Witwatersrand, Johannesburg, South Africa. With a foreword by Sir Arthur Keith. (Thesis approved for the D.D.S. degree in the University of the Witwatersrand.) 1931: pp. 134; 7½ x 4½ in.; 54 illustrations; 12s. 6d. John Bale, Sons and Danielsson, Ltd., Oxford House, 83 Gt. Titchfield St., London, W. 1, England.

## INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

*Meetings in 1930–31*

SECTIONS. *Ann Arbor:* June 11. *Boston:* January 26, May 14. *Chicago:* October 16, January 8, February 1, April 30. *Cleveland:* March 9. *Halifax:* November 28, February 28, April 24. *London:* June 9. *Minnesota:* October 27, November 24, January 26, March 30, April 27. *New Haven:* February 12. *New York:* December 10, May 21. *Philadelphia:* October 13, June 24. *Pittsburgh:* November 3, December 1, January 5, February 2, March 2, May 18. *St. Louis:* December 23, January 13, April 14. *Toronto:* February 5.

ASSOCIATION: Ninth general meeting, Memphis, March 21–22, 1931.

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